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Editorial

The Lecture Room and Note Taking

We have always felt badly for the professor who lectures away to a multitude of bowed heads of students whose only interest seems to be in getting down in black and white his every word. This may be a sort of compliment to the teacher, whose words, as it were, are pearls not to be esteemed lightly, but while doing this, much of the context of the thought is missed, and the forest cannot be seen for the trees.

Lectures are supposed to be a stimulus for thought, not a grand and complete summary of the whole field. Most men of sense do not presume to be the last authority on their subject, and we must realize that there are antagonistic views which may be the correct ones. When we routinely copy a lecture, and cram it up for exams, having done no thinking whatsoever, we are doing nothing more than any high school student might do. It is granted that notes are a great aid in passing exams; some are set entirely on them—unwisely so—for the man who sets such an examination, and expects a routine answer, is really bigoted in himself and in his knowledge, and is running contrary to the first law of education—freedom of thought.

There are many things in medicine that must be learned, must be solidly memorized, but all of them are standard, and can be found in any text-book of good quality. If we write down a line here and there in the lecture, it impresses the material in our minds, but if we copy down everything, the impression is nil,

and we have the dickens of a time sorting the wheat from the chaff later on. Far better is it to take no notes at all, and just listen, than to be such a robot.

What's one man's meat is often another man's poison, and there is no set rule in note taking to be followed by all of us. But if we must take notes, and most of us like to take a few, we should accustom ourselves to record the pertinent facts as briefly as possible. The lecturer would rather see a face than a head of hair, and the time spent in note taking should be a portion of the time spent in listening. The real teacher would rather stimulate a lively curiosity and outside reading, than have his own ideas swallowed "holos bolus" with never a particle of thought on them.

It is true that with our multitude of subjects, our outside activities both social and otherwise, there is not a great amount of time to spend reading text-books, yet one can often read a subject over in a text-book as fast as one can decipher one's lecture notes, hurriedly and frantically scribbled in the classroom for fear of missing something. And what if we do miss something in the lecture room that is on the exam—it makes little difference in the scheme of things whether we make a 60 or a 65—but it does make a difference to us if our knowledge is gained by ourselves, and not a product of mass spoon feeding.

In the clinical years, lectures and lecture notes should take purely a secondary place to clinics, and if we have to choose between reading up on a clinic or reading up on a lecture—through sparsity of time—we should take the former; for the subject matter will be more alive to us when we have seen the patient, and one picture is worth a thousand words.

THE TYPES OF INTRACRANIAL HAEMORRHAGE CAUSED BY HEAD INJURY

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Introduction

The most important cause of damage to the intracranial structures in head injuries is haemorrhage and the pathologist's main interest, therefore, is the study of the types of haemorrhage which may be thus produced.

Fracture of the skull will not be dealt with in this article. It is naturally present in the majority of cases showing severe intracranial haemorrhage, but it is of great importance to remember that haemorrhage may be sufficiently severe to cause death rapidly without an accompanying skull fracture. Fractures of the base of the skull are of particular significance if the fracture line runs through the cribriform plate of the ethmoid, the accessory nasal air sinuses or the middle ear cavities. The danger in such cases is the provision of a pathway by which micro-organisms may reach the meninges to cause a fatal meningitis. Meningitis as a complication of head injury has, therefore, to be borne in mind, although it does not come within the scope of this paper.

Extradural Haemorrhage

This is, as a rule, caused by a blow on the side of the head sufficiently severe to fracture the thin squamous portion of the temporal bone. On the other hand fracture is not necessarily present in this type of intracranial haemorrhage which may kill the patient within a few hours of his injury.

The haemorrhage typically occurs from rupture of the anterior division of the middle meningeal artery on the side of the head which received the blow. The effused blood is under arterial blood-pressure and, therefore, rapidly strips up the dura mater to form an extradural haematoma. When this arterial haematoma has become thick enough to compress the underlying cerebral cortex the lesion may be localized by the appearance of signs of paralysis of the opposite side of the body. The oculomotor nerve on the side of the haemorrhage is frequently stretched as it runs forward immediately below the hippocampal uncus of the compressed hemisphere, causing dilatation of the pupil on the side of the haematoma.

Immediately a diagnosis is made it is necessary to evacuate the blood-clot by surgical operation and to stop further haemorrhage from the ruptured middle meningeal artery, which should give good results in uncomplicated cases.

One of the difficulties in the diagnosis of this type of haemorrhage is that there is not infrequently a "lucid interval" between the transitory unconsciousness at the time of the injury and the evidence of compression of the brain by the haemorrhage. In this "lucid interval" the patient may be apprehended by the police and locked up as a probable "drunk". In cases of difficult differential diagnosis between "drunk" and "dying" the possibility has always to be borne in mind that a man may be drunk and dying of a middle meningeal haemorrhage.

A good illustrative case showing the danger of the lucid interval and the urgency of this type of haemorrhage is that of a young man who hit the side of his head on a metal post about 11.00 p.m. while skating. He was stunned by the blow but was able to go home and go to bed with no suspicion that the injury had been a serious one. Two or three hours after he had gone to bed his parents noticed him to be breathing heavily. A doctor was called at once, who found him unconscious. He was rushed to hospital but died about 6.00 a.m., seven hours after his injury, before an operation could be performed. Post-mortem examination showed a typical, uncomplicated middle meningeal haemorrhage.

The main complication of extradural haemorrhage from the pathologist's point-

of-view is that the blow may have been sufficiently severe to cause laceration of and haemorrhage into the cortex of the opposite cerebral hemisphere, (contre-coup laceration), with haemorrhage into the subarachnoid space. Hence, cases of middle meningeal haemorrhage with this complication will be likely to show blood in the cerebrospinal fluid which the uncomplicated cases will not. The prognosis in cases showing blood in the cerebrospinal fluid will, naturally, not be so good as those in which the diagnostic lumbar puncture fluid is clear.

Subdural Haemorrhage

This is venous haemorrhage, the blood as a rule escaping into the subdural space over the convexity of one or both cerebral hemispheres. In the great majority of cases the haemorrhage comes from a ruptured superior cerebral vein at the vertex of the hemisphere where the vein is passing through the subdural space to pour its blood into the superior sagittal sinus.

The injury which produces this haemorrhage is, typically, a slight one and in a considerable percentage of cases no history of even a minor bump on the head may be obtained. The blow tends to be to the front or the back of the head, displacing the cerebral hemispheres backwards or forwards enough to tear the vein at or near its fixed attachment to the wall of the sinus. A bump against a furnace-door or a beam may be sufficient to rupture one of these thin-walled veins. In the latest case recorded in our surgical files the patient's injury was a blow on the forehead received while he was "boxing" with his young son.

It is obvious that such slight injuries as these do not fracture the skull and the patient complains of nothing more than headache immediately following the accident. This headache may persist as the blood, at low venous pressure, is slowly effused into the subdural space. This "space" is normally a very narrow interval between the inner endothelial lining of the dura and the outer endothelial lining of the arachnoid. There are very few fibrous septa connecting these two membranes and consequently the blood in the subdural space can widen the interval into a space by slowly compressing the arachnoid and the underlying brain. As the blood is leaking into an endothelial-lined space, with little tissue-damage to liberate thrombokinase, clotting is slow and the blood as a rule spreads widely over the whole of the convex surface of the hemisphere. The further spread of the subdural haemorrhage becomes limited by the formation of a membrane around it to form a haematoma. The outer membrane of this haematoma is in intimate contact with the inner surface of the dura mater, so much so that some authorities believe that it is produced from this membrane. The inner membrane of the haematoma is thinner than the outer and is not adherent to the underlying, compressed arachnoid membrane.

The formation of this well-named "chronic traumatic subdural haematoma" is, therefore, slow, as opposed to the extremely rapid formation of the arterial haematoma of middle meningeal haemorrhage. As a rule compression of the underlying cortex by the haematoma is not sufficiently severe to produce localizing signs until some weeks have elapsed since the injury. In a series of cases recently reviewed, the average length of time between the injury and operative treatment was about four months.

Operative treatment consists of draining the haematoma through a suitably-placed burr-hole in the skull. The diagnosis can often be confirmed before the dura mater is opened, as the underlying blood gives a bluish colour to the exposed membrane. Drainage of the haematoma through a small burr-hole is facilitated by the contents of the haematoma being a mixture of fluid blood and small poorly-formed clots, which can be reasonably easily washed out. The results of this simple operative procedure are satisfactory.

The diagnosis of subdural haematoma may be very difficult, especially if there is no known history of injury or if lateralizing or localizing signs are confusing or absent. One result of this has been that the condition may not be suspected and, especially if haematomata are compress-

ing both frontal lobes, the patient may show mental deterioration sufficient to necessitate his admission to a mental hospital. Old subdural haematomata, therefore, are found with considerable frequency in post-mortem examinations of mental hospital patients. One of our cases attempted suicide by a revolver shot. The bullet produced a comminuted fracture of the frontal bone. An operative exploration to remove bone fragments revealed an old subdural haematoma under the recent fracture of the skull. This haematoma was caused by a head-injury eleven years before, following which there had been progressive mental deterioration. The patient's mental state has improved since the removal of this old subdural haematoma.

Infection of a subdural haematoma is a rare complication in civilian practice. Infection of the subdural space is an extremely serious condition which is almost invariably fatal in spite of heroic surgical measures for its cure.

In view of the important late complications of chronic traumatic subdural haematoma and of the difficulties of its diagnosis, its presence is being suspected in an increasingly large number of cases. If there is a reasonable suspicion that this condition may be the cause of the patient's symptoms, bilateral exploratory burr-holes provide a simple way of confirming or dispelling the suspicion.

Subarachnoid Haemorrhage

It is obvious that any head-injury sufficiently severe to damage the cerebral cortex will cause haemorrhage into the subarachnoid space. Fractures of the skull may damage vessels in the subarachnoid space causing haemorrhage. Trauma produces subarachnoid haemorrhage as an event secondary to damage of surrounding structures and primary uncomplicated traumatic subarachnoid haemorrhage is very rare.

Spontaneous subarachnoid haemorrhage may be honestly thought by the patient to be caused by a head injury while at work. In one of our cases the patient was employed chipping stones when he suddenly suffered a severe pain in the head, which he naturally attributed to a flying piece of stone. On examination there was no evidence of head injury. There were large quantities of blood in his cerebrospinal fluid and he rapidly developed a hemiplegia. He died about 36 hours after the onset of the pain in the head. Post-mortem showed the sudden pain and the subsequent hemiplegia to be due to rupture of an aneurysm of the middle cerebral artery of the side opposite to the hemiplegia.

Cortical Haemorrhage

Traumatic haemorrhage into the cerebral cortex may, of course, be produced by direct laceration of the cortex by depressed fragments of bone in comminuted fractures of the skull, or by penetrating wounds of the skull by bullets, shell fragments or other foreign bodies.

In the usual civilian head injury it is comparatively rare to find cortical laceration and haemorrhage below the area of the skull which has received the blow. It is, however, very usual to find contre-coup cortical laceration and haemorrhage of the parts of the brain diametrically opposite to the point of impact of the blow. Here again, as in cases of extradural and of subdural haemorrhage, severe contre-coup cortical laceration may be seen in cases where there is no fracture of the skull. This type of injury may be found in any cortical area depending on the site of the traumatizing force, but it is seen most typically involving the tips of the frontal and temporal poles of the brain as the result of a blow on the back of the head.

Anatomically, contre-coup laceration usually is seen as an area of considerable extent in which the cortex is mottled with small haemorrhages. In the more severe cases these intracortical haemorrhages have ruptured through the pia-mater into the subarachnoid space causing one of the types of secondary subarachnoid haemorrhage. As mentioned above, this is the usual reason for the finding of blood in the lumbar puncture fluid in a case of extradural haemorrhage.

An interesting case showing typical frontal and temporal contre-coup haemorrhage at post-mortem was that of a

woman who was found unconscious at the bottom of the steps of her house early one morning. Additional findings were a severe fracture of the side of the occipital bone opposite to the more marked frontal cortical laceration, and thrombosis of the basilar artery. From these findings it was presumed that the basilar thrombosis had caused her to fall, striking the back of her head and producing severe contre-coup cortical laceration and haemorrhage. There was no direct cortical laceration underlying her fractured occipital bone.

Attempts at operative treatment of this type of cortical injury are unsatisfactory as the cortical tissue is, as a rule, a pulped haemorrhagic mass in which satisfactory haemostasis is difficult to obtain.

Cases with severe contre-coup cortical laceration may, however, recover from the immediate effects of their head injury, the old traumatized cortical tissue being uncovered at post-mortem examination many years after the head injury. In such cases, perhaps particularly in those in which the trauma has destroyed considerable areas of the frontal cortical tissue, gradual mental deterioration from the time of the injury is a not uncommon clinical story.

A second important sequela of cortical injury is traumatic epilepsy. This, as a rule, begins some years after the receipt of the head injury. It is usually found in cases which have had a depressed, comminuted fracture of the skull, directly underlying which severely scarred, gliosed cortical tissue is found at operation or post-mortem. The epileptic seizures may show lateralizing signs corresponding with the hemisphere which contains the scarred cortical area, or the epilepsy may be generalized.

Wide surgical excision of the scar, removing cortex and white matter down to, and opening into, the cavity of the lateral ventricle gives satisfactory results with alleviation of the epilepsy in some cases.

Deep Intracerebral Haemorrhage

Petechial haemorrhages in the deep tissues of the cerebral hemispheres are of some importance as an index of concussion of the brain, but massive haemorrhage into the white matter or into the capsular region of the brain is practically never caused primarily by head injury.

Anomalies of the development of blood-vessels to form blood-vessel "tumours" or varices are not uncommon incidental findings in the white matter and, if a person who has one of these "tumours" suffers a head injury, the injury may produce massive haemorrhage from the tumour.

Massive haemorrhages into the external or internal capsules are considered to be spontaneous rupture of the responsible vessels and, when a capsular haemorrhage is associated with a history of head injury, the argument is generally upheld that the capsular haemorrhage was the cause of the accident. A man drove his motor-car into a standard in the middle of the street and died of severe intracranial injuries within a few minutes. He was found at post-mortem examination to have a capsular haemorrhage, which was believed to have preceded and to have been the cause of his accident.

Midbrain Haemorrhage

The midbrain is intimately surrounded on its dorsal and lateral sides by the free inner edge of the tentorium cerebelli. It is conical, with the base of the cone above, so that pressure from above tends to wedge the midbrain more firmly in the tentorial "hiatus", as well as causing traction on the oculomotor nerves at the point where they leave the ventral surface of the midbrain.

The effects of trauma to the midbrain in cases of severe head injury are mainly suggested by clinical evidence of damage to one or both oculomotor nerves, or of damage to the corticospinal tracts which lie in the superficial tissue of the cerebral peduncles and may be bruised by pressure against the free edge of the tentorium cerebelli.

With reference to the first of these effects, one of our cases had one oculomotor nerve torn out of the midbrain. Internal traumatic haemorrhage in the midbrain not infrequently involves the oculomotor nuclei or the axones of the cells of these nuclei as they run ventrally

through the midbrain tissue. One of the sequelae of a severe head injury may therefore be paralysis of the oculomotor nerve on one or both sides. In one case the midbrain haemorrhage was contre-coup in situation, being more marked in the oculomotor nucleus region of the side opposite to the injury. This suggested lateral dislocation to the upper brain-stem away from the point of impact and against the edge of the tentorium on the opposite side.

With such a mechanism it follows that the corticospinal tract is more vulnerable on the side of the midbrain opposite to the direction of the force applied and, therefore, that such cases may show the "false lateralizing" sign of a hemiplegia of the same side as that receiving the blow.

Pontine Haemorrhage

Pontine haemorrhage is, naturally, a very grave complication of head injury and, presumably, is always rapidly fatal.

The mechanism of its production is obscure. It may be caused by a forcible downward thrust of the brain-stem when the intracranial pressure above the tentorium is suddenly and violently raised by the infliction of the blow. This downward thrust may be sufficiently severe to cause impact of the pons against the basi-occipital bone, with consequent bruising of the pontine tissue. In the most severe cases the laceration of the brain-stem may extend throughout the midbrain and pontine tissues. In these latter cases the patients do not survive the head injury for more than a few hours.

They may be found at post-mortem examination to explain rapid and unexpected death following a head injury. An example is that of a man who fell downstairs while carrying a heavy box. He was unconscious for fifteen minutes, had a short lucid interval and then relapsed into unconsciousness. His left pupil was dilating. With these findings a diagnosis of left middle meningeal haemorrhage was made. This was confirmed and successfully treated by operation two hours after his accident. He remained comatose and died with a rising pulse and temperature and a falling blood-pressure, nine and a half hours after his accident. Post-mortem examination showed the artery to have been ruptured in a fracture-line at the outer angle of the left lesser wing of the sphenoid. The brain was severely oedematous. The brain-stem contained streaky traumatic haemorrhages in the midbrain and pontine tissues.

It follows from the above that the brain-stem must be examined in post-mortem investigation of every case of head injury.

Summary

(1) The more common types of traumatic intracranial haemorrhage have been reviewed and have been illustrated by brief case-reports.

(2) Some of the more important sequelae of intracranial traumata have also been discussed.

The material used in this paper has been collected in part from the Neurological Surgical Service of the Toronto General Hospital and I am grateful to Dr. McKenzie and to Dr. Botterell for the opportunity of studying their cases.

I am also grateful to Dr. W. L. Robinson for his kindness in allowing me to investigate the material which he receives as a Coroner's Pathologist for the City of Toronto.

ACIDOSIS AND ALKALOSIS IN INFANCY AND CHILDHOOD

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For the maintenance of life the reaction of the inner body fluids must be slightly alkaline. Normally the reaction lies near pH 7.4. The extreme ranges which have been observed in the human are from pH 7.0 to 7.8. The factors involved in the maintenance of a correct body fluid hydrogen ion content include the balance of bases and acids in the body fluids, the excretion of carbonic acid by the lungs, and the excretion of bases and acids by the kidneys.

The two immediate factors of control of extracellular fluid reaction are the concentrations of the acid free carbonic acid and the alkaline base bicarbonate. A ratio of 1 part of carbonic acid to 20 parts of bicarbonate, under normal conditions 3 volumes per cent free CO_2 and 60 volumes per cent as bicarbonate, produces a reaction of pH 7.4.

It will be seen that disturbances in the $\frac{1}{20}$ ratio between free CO_2 and bicarbonate can be produced by a change in concentration of either of these substances, with no change in the other. With the bicarbonate remaining stationary, an increase in plasma free CO_2 would produce an acidosis, a decrease in CO_2 an alkalosis. However, the concentration of free CO_2 in the plasma is controlled by the tension of CO_2 in the residual air in the lung alveoli to which the plasma is exposed as it passes through the alveolar capillaries. So excellent is this respiratory control of plasma free CO_2 that we rarely encounter changes in the reaction of blood plasma due to primary changes of CO_2 content. The deviations from the normal value pH 7.4 of the plasma are therefore practically always due to changes in its bicarbonate content.

Hence in practically all clinical conditions acidosis may be defined as a condition where the amount of bicarbonate in the blood stream is decreased, and alkalosis a condition in which blood bicarbonate is increased. The bicarbonate content of blood serum can be determined easily. The blood is collected and centrifuged under oil, the serum delivered into a gas analysis apparatus where the amount of CO_2 freed by the addition of acid is measured under standard pressure and temperature. Normally this ranges from 55 to 60 c.c. of CO_2 per 100 c.c. serum or 27 c.c. $\frac{N}{10}$ bicarbonate per 100 c.c. serum.

The changes which occur in the bicarbonate content of the serum, however, are not primary changes but are the result of changes in the other members of the acid base system of the blood. Normally 100 c.c. blood serum contains 157 c.c. $\frac{N}{10}$ fixed base, largely Na. (132 c.c.) with smaller amounts of K., Ca. and Mg. At the reaction of blood serum these amounts of fixed base are exactly covered by acids, largely Cl (103 c.c.), bicarbonate (27 c.c.), and smaller amounts of phosphate, sulphate, organic acid such as the ketone acids acetoacetic and B-oxybutyric; and protein. Of these acids bicarbonate is the weakest, hence the amount of bicarbonate present in the serum is equal to the sum of the fixed bases present minus the sum of the acids stronger than bicarbonate. For comparison both acid and basic substances must be expressed in milliequivalents per liter or c.c. $\frac{N}{10}$ per 100 c.c.

If with fixed base remaining normal there is an accumulation of these stronger acids, bicarbonate is displaced with freeing of carbonic acid, and this carbonic acid is excreted through respiration. If through loss of the stronger acids from the blood fixed base is uncovered, the carbonic acid present in the blood immediately unites with the free base to produce exact balancing of the bases and acids by an increase in bicarbonate. Where the amount of

fixed base is decreased the total amount of acids is equally decreased at the expense of the weakest acid, namely, bicarbonate.

When an acidosis is produced by a lowering of bicarbonate, the body attempts to restore the $\frac{20}{1}$ ratio between bicarbonate and carbonic acid and thus maintain a normal reaction by decreasing the amount of free carbonic acid in the blood. This is brought about by decreasing alveolar $CO_2 = CO_2$ by *deep pauseless breathing*, the typical breathing of acidosis. When blood bicarbonate is increased (alkalosis) the free carbonic acid is increased by *slow shallow respirations* in an attempt to maintain the normal ratio. Were this compensatory respiratory reaction functioning absolutely correctly, no changes in reaction would be brought about by changes in the bicarbonate. However, the mechanism is not absolutely perfect, hence where bicarbonate is decreased the reaction of the blood becomes slightly more acid than normal, and with increasing bicarbonate, slightly more alkaline. These changes from normal, however, are not nearly as marked as would be the case if the safety factor of changes in ventilation did not occur.

In attempting to make use of this change in ventilation in the diagnosis of acidosis or alkalosis, one should be careful to make observations of rate and depth of respiration when the patient is undisturbed. The author well remembers a case of alkalosis which when undisturbed breathed at the rate of 5 per minute. However, before the chief of clinic was due to visit, the patient was carefully "rearranged and made comfortable", with a consequent disturbance sufficient to change the respiratory rate to approximately normal.

Through filtration and reabsorption and the ability to excrete urine at a reaction differing from the blood, the kidneys play a large part in maintaining the concentration and balance of the bases and acids of extracellular fluid. However, for kidney function to be efficient, water must be available. In the normally hydrated body when sodium chloride is administered either sodium or chloride ions can be retained and the unwanted ions, either basic or acid, can be excreted. However, in the dehydrated infant if isotonic saline solution is administered both Na and Cl must be retained in the proportions in which they are present. Since the ratio of $\frac{Cl}{Na}$ is $\frac{103}{132}$ in serum and $\frac{154}{154}$ in normal saline, this increases the acidosis.

Treatment

In the treatment of acidosis and alkalosis, 5% glucose solution, normal saline solution, or mixtures of the two are the substances most frequently administered parenterally. Glucose solution in addition to repairing ketosis restores renal function by re-establishing plasma volume and providing adequate volume flow of blood through the kidneys. The glucose is soon oxidized, providing surplus water to be removed by the kidneys, which facilitates the selective excretion of either acids or bases from the blood. No advantage is obtained through the use of Ringer's solution instead of normal saline for repair of the acid base structure. Normal saline supplies Na and Cl. The small requirements of K, Ca and Mg for repair of extracellular fluids are available through the metabolic processes which carry on even in starvation.

In exceedingly severe acidosis it may be necessary to increase the plasma bicarbonate immediately. In these cases one may use a 4% soda bicarbonate solution or a sodium lactate mixture, the lactate portion of which is destroyed in metabolism and replaced by bicarbonate. It is important to remember that dehydration is always encountered along with acidosis and alkalosis; hence we must restore the volume as well as the structure of the extracellular fluid.

In the preparation of glucose solutions, only grades of dextrose manufactured specially for intravenous use may be used. A solution of the required strength is prepared with freshly distilled water filtered through No. 50 Whatman filter paper and sterilized for

20 minutes at 10 pounds pressure. We have not, in the Hospital for Sick Children, experienced temperature reactions after either subcutaneous or intravenous injections of glucose. If reactions are encountered due to pyrogenic substances, these may be removed by the use of a seitz filter. Normal saline solution is prepared by dissolving 9 grams of "Reagent" sodium chloride in 1 liter of freshly distilled water, filtering and sterilizing for 30 minutes. Mixtures of 2 parts 5% glucose solution with 1 part normal saline solution may be made up and then sterilized for 30 minutes. In the Hospital for Sick Children, glucose solutions prepared in the Hospital are not used when they are more than 3 days old.

Solutions of 4% sodium bicarbonate cannot be sterilized by heating without the formation of the irritating carbonate. Bicarbonate solutions may be prepared aseptically. With a sterile spatula the bicarbonate is removed from the centre of a fresh container of the salt, weighed into a sterile container to which is added cooled sterilized freshly distilled water.

Sterile molar sodium r-lactate may be obtained in stable solution in ampules or may be prepared in the laboratory. From this may be prepared a mixture of 1 part isotonic sodium lactate solution to 5 parts isotonic sodium chloride solution, the mixture then being sterilized as for normal saline solution.

Stable sterilized solutions for parenteral use may be obtained from various manufacturers if desired, with a sterile setup for administration.

Fluids may be administered parenterally by either the subcutaneous, intravenous or intraperitoneal routes. The subcutaneous route is the easiest and safest. By this route one may administer approximately 10 c.c. of fluid, saline or saline lactate solutions at one injection. Subcutaneous injections may be administered 1, 2 or 3 times daily, depending upon the degree of dehydration and the rate of absorption. In infancy and childhood less discomfort is experienced if the site of injection is the upper abdomen, just below the rib margin, or in the back if one wishes to examine the abdomen as in a suspect pyloric stenosis, rather than in the axilla. The solution for injection is warmed to about 100° F, and temperature is tested by allowing a few drops to run onto the wrist and the injecting apparatus is filled. The container is suspended or held about 2 feet above the patient and the injecting system is freed from air by allowing fluid to run out and placing a clamp on the rubber tubing. Injection of a small amount of air will do no harm to the patient, but when the stethescope bell is placed over an area of subcutaneous emphysema, with movement very fine cracklings will be heard which occasionally are confused with sounds produced in the lung itself. The patient is restrained by wrapping, the skin is prepared with 70% alcohol, a fold of the skin is picked up, and the needles are inserted in the subcutaneous tissue upwardly, parallel to the skin for a distance of 1 or $1\frac{1}{2}$ inches, and the fluid is allowed to run in by gravity. When the injection is completed, the tubing is clamped, the needles removed, and the puncture wounds are sealed by a bit of sterile absorbent cotton and painted over with collodion solution.

Intravenously, fresh or citrated whole blood, glucose, saline, bicarbonate or lactate solutions may be administered. In the infant, a single injection may consist of from 12 to 15 c.c. per pound of body weight. In the older child, from 250 to 400 c.c. may be administered. A single injection may be given into any of the superficial veins, median basilic, external jugular or, in the tiny infant, the superficial scalp veins. Injections of any fluid into the superior longitudinal sinus through the open anterior fontanelle are not devoid of danger and this route should never be used for injection of any but clear, non irritating, fluids on account of brain damage which may result if the sinus is transfixed.

By continuous intravenous injection, from 125 to 150 c.c. in the infant may be injected per 12 hour period. Occasionally in the older child the needle may be inserted directly into the vein and held in place, but (and certainly in infancy) in most children one should cut

down on a vein, usually the internal saphenous just anterior to the internal malleolus, and tie a needle into the vein. Where a transfusion has been given, the continuous intravenous apparatus can be set up in the operating room and the patient returned to the ward with the solution running. The continuous intravenous will usually run for 2 or 3 days before stopping. At the least sign of any phlebitis it should be discontinued.

In our hands intraperitoneal injection has not been without danger. Certainly this route should not be used when abdominal distension is present and only saline or saline lactate solutions may be administered.

In the treatment of acid base disturbances—(1) sugar solution, (2) citrate, orange juice and glucose solution (C.O.G.), and (3) orange juice, sugar and water solution may be given by mouth. Fifteen per cent sugar solution, half dextri-maltose and half either glucose, lactose or cane sugar, supplies calories and antiketogenic substances in addition to water. C.O.G. solution consists of 1 part of 1% sodium citrate solution, 2 parts of orange juice, and 3 parts of 10% glucose solution. This solution in addition to supplying calories, antiketogenic substances and water also counteracts acidosis, due to the fact that in the process of metabolism the organic acids are destroyed and the base left uncovered to be bound as bicarbonate. Equal parts of orange juice and water sweetened with cane sugar may take the place of C.O.G. in the home care of the infant. Where vomiting is present, these fluids are more apt to be retained if administered cold, at first in teaspoonful doses at 5 minute intervals. As vomiting subsides the amount administered at one time is increased and the interval is lengthened.

On account of lack of cooperation by the patient, rectal administration of fluid is impracticable in infancy, and of course this route is ruled out in diarrhoea, *which is the cause of most acid base disturbances in infancy and childhood.*

Acid base changes in acute intoxication due to enteral or parenteral infection.

Here we have a decrease in bicarbonate, an acidosis, due to (a) loss of bases through excessive excretion of alkaline intestinal secretions, (b) increased concentration of phosphates and sulfates through defective kidney excretion, since the amount of water available for kidney function is lowered, and (c) if food is persistently vomited, an accumulation of ketone acids. In about 60% of cases the chlorides are lowered due to the vomiting, but this lowering of acid is not sufficient to produce a normal bicarbonate due to the changes in the other ions both basic and acid.

The treatment of this condition is the following:

(1) Transfusion of whole blood.
(2) Following the transfusion, through the same needle used for the transfusion, a continuous intravenous of 2 parts 5% glucose solution, 1 part normal saline solution, is started before the infant is returned to the ward.

When this continuous intravenous ceases to run, it may be necessary to restart it, since the dehydration may have been repaired to such a degree that further administration of fluid parenterally may be unnecessary, or it may be administered by the simpler subcutaneous route. By mouth citrate, orange juice and glucose, or a similar solution, should be administered. When the infant has been free from toxicity for about 48 hours and the vomiting has ceased or is only occasional, the infant may be started on weak dilutions of protein or evaporated milk.

Other Clinical Examples

In *inanition fever* of the newborn, the acidosis due to formation of ketone acids through starvation can be usually corrected by oral administration of sugar solution. If the condition is severe, subcutaneous administration of 5% glucose solution or $\frac{2}{3}$ 5% glucose solution, $\frac{1}{3}$ normal saline solution may be necessary.

The *acidosis of weaning*, due to the formation of ketone acids through persistent refusal of the artificial feeding, should never occur. From an early age the breast fed infant should be accustomed to take water from the bottle and solid foods should be introduced into its diet at the proper time. If through mismanagement acidosis occurs, the condition should be treated by intravenous or subcutaneous injection of 5% glucose, depending upon the severity of the condition.

In the treatment of *diabetic acidosis* the main therapeutic procedure is the destruction of ketone acids by insulin in the presence of glucose. As the ketone acids are destroyed the fixed base uncovered unites with carbonic acid to increase the bicarbonate content. At the same time dehydration may be combatted by the use of normal saline solution parenterally. In profound acidosis the immediate use of 4% bicarbonate solution or a mixture of 1 part isotonic sodium lactate solution to 5 parts of isotonic salt solution may be necessary as a life saving measure. When these solutions are used, it must be remembered that symptomatic improvement of the acidosis may result before relief of the ketosis, cure of which can only be judged by the disappearance of acetone bodies from the urine.

Alkalosis, an increase in bicarbonate, is encountered clinically only in cases of pyloric stenosis, with subsequent loss of HCl through vomiting of gastric secretion. Where the pyloric obstruction is incomplete, as in congenital hypertrophic pyloric stenosis, and only a portion of the gastric secretion is lost, a clinical alkalosis may take weeks to develop. Where complete obstruction is present, marked alkalosis will develop in 2 or 3 days. It should be noted that in experimental complete pyloric obstruction in rats, $\frac{1}{3}$ of all the chloride in the body may be lost in the short period of 12 hours. Where obstruction is complete, a ketosis is also present, due to carbohydrate starvation. However, the loss of chloride is so great, and such a large amount of base is uncovered by this loss that the net result is an increase in bicarbonate (alkalosis) in spite of the ketosis. Here we have an alkalosis plus a ketosis, with appearance of ketone bodies in the urine. In fact the alkalosis would be still more marked if a portion of fixed base were not covered by ketone acids.

For the treatment of alkalosis, the solution administered parenterally should be normal saline solution to which 5 grams of glucose per 100 c.c. of solution has been added. Through metabolism of the glucose the ketosis is repaired. The increased concentration of Cl in this solution will repair the chloride loss more rapidly than would be a mixture of 2 parts of 5% glucose and 1 part of normal saline.

"Halitosis Impairs the Organ of Hearing!"

"Instances are on record in which very baneful and injurious effects have resulted from the practise of speaking into the ear, more especially where the breath of the person is tainted. One case I may mention is related by Lord Herbert. Cardinal Wolsey, he tells us, towards the latter part of his life, was in the habit of whispering into the ear of his sovereign Henry VIII; and the serious indisposition of the King has been many times attributed to this cause, and certainly not without reason."

A treatise on the Physiology and Pathology
of the Ear.
By John Harrison Curtis.
London MDCCCXXXI.

ACUTE OTITIS MEDIA

By JOSEPH A. SULLIVAN, M.B.

Department of Otolaryngology, University of Toronto and St. Michael's Hospital

Along with the common cold and dental caries, some type of otitis media occurs, at one time or another, to nearly every individual. It is paramount that one immediately realizes that acute otitis media is often very commonly the precursor of mastoiditis, petrositis, labyrinthitis, Bezolds Abscess (perforation of the mastoid tip producing an abscess in the neck), sigmoid sinus thrombosis, brain abscess, and finally meningitis. There are many who are never aware of it, but examination of pathologic specimens almost invariably shows evidence of scar tissue in some part of the middle ear, and these telltale tracks show why people, as they grow older, usually lose a certain amount of their hearing for air conduction sounds.

The diagnosis and treatment of diseases of the middle ear may be extremely difficult, because the cavity lies deep inside the skull and often ramifies into extensive or hidden mastoid and petrosal air spaces. Nature, however, has provided us with a semitransparent window, called the ear drum or tympanic membrane, through which one can obtain some information as to what is going on inside the middle ear. It is this window which is examined so carefully when the patient comes in complaining of symptoms which suggest ear disease. Unfortunately the window is very small and does not always give a true picture of what is going on behind it. It is necessary, therefore, for the clinician to bring all available information to his aid.

Anatomy and Embryology

To understand otitis media one should know something of the development and detailed anatomy of the temporal bone. This cranial bone which contains the middle ear, is undoubtedly the most complicated structure in the body. It consists of relatively flat outer parts called the squamous and mastoid portions from which projects inward a roughly three sided structure called the petrous pyramid. This pyramid is the most important part of the temporal bone because it forms the keystone of the base of the skull and contains the middle ear and labyrinth. The labyrinth will not concern us in this paper because it is only secondarily involved in infections of the temporal bone. Emphasis will be placed on the middle ear because it is the primary focus and the most important drainage point. The petrosa measures approximately one inch in infants and anywhere from 2½ to 3 inches in adults.

The middle ear, tympanum, or tympanic cavity may be described roughly as a wedge-shaped cavity containing the three small bones called the malleus, incus and stapes respectively. It is divided into three portions—(Figs. 1, 2).

1. *The epitympanic space or attic*—which is that part of the tympanic cavity which lies above the level of the short process of the malleus.
2. *The atrium or mesotympanic space*—which is that portion of the middle ear cavity which lies below the short process of the malleus and above the floor of the bony external canal.
3. *Hypotympanic space*—that portion of the middle ear which lies below the level of the floor of the bony canal.

Suffice is it to say in this presentation that the tympanic cavity has five walls, and tapers anteriorly into the eustachian tube which drains into the nasopharynx. Simply stated, the external auditory meatus develops from the first branchial cleft, while the middle ear and eustachian tube develop from the first branchial pouch. The lining of the middle ear cavity, therefore, is made up of mesoderm and entoderm which is continuous with that of the nasopharynx. The ear drum is that partition between the outside and inside invagination and consists therefore, not only of the lining of the middle ear but also of the stratified squamous epithelium which develops from the ectoderm of the embryo. Without going into

detail it makes a great difference whether the temporal bone is highly pneumatized or not. The great surface presented by many air cells lined with mucous membrane can excrete entirely too much fluid, if inflamed, to evacuate through the eustachian tube or through a perforation in the drum membrane. Extension of the inflammation and pain due to the pressure are the result. Furthermore, if the ear because air cells in and around it vary so tremendously from case to case. When the middle ear becomes infected, these air cells also become infected so that it is impossible to have otitis media without some mastoiditis, and if the petrosa is pneumatized, impossible to have otitis media without some petrositis. In my opinion it is really a wonder that infections of the mastoid so often get well

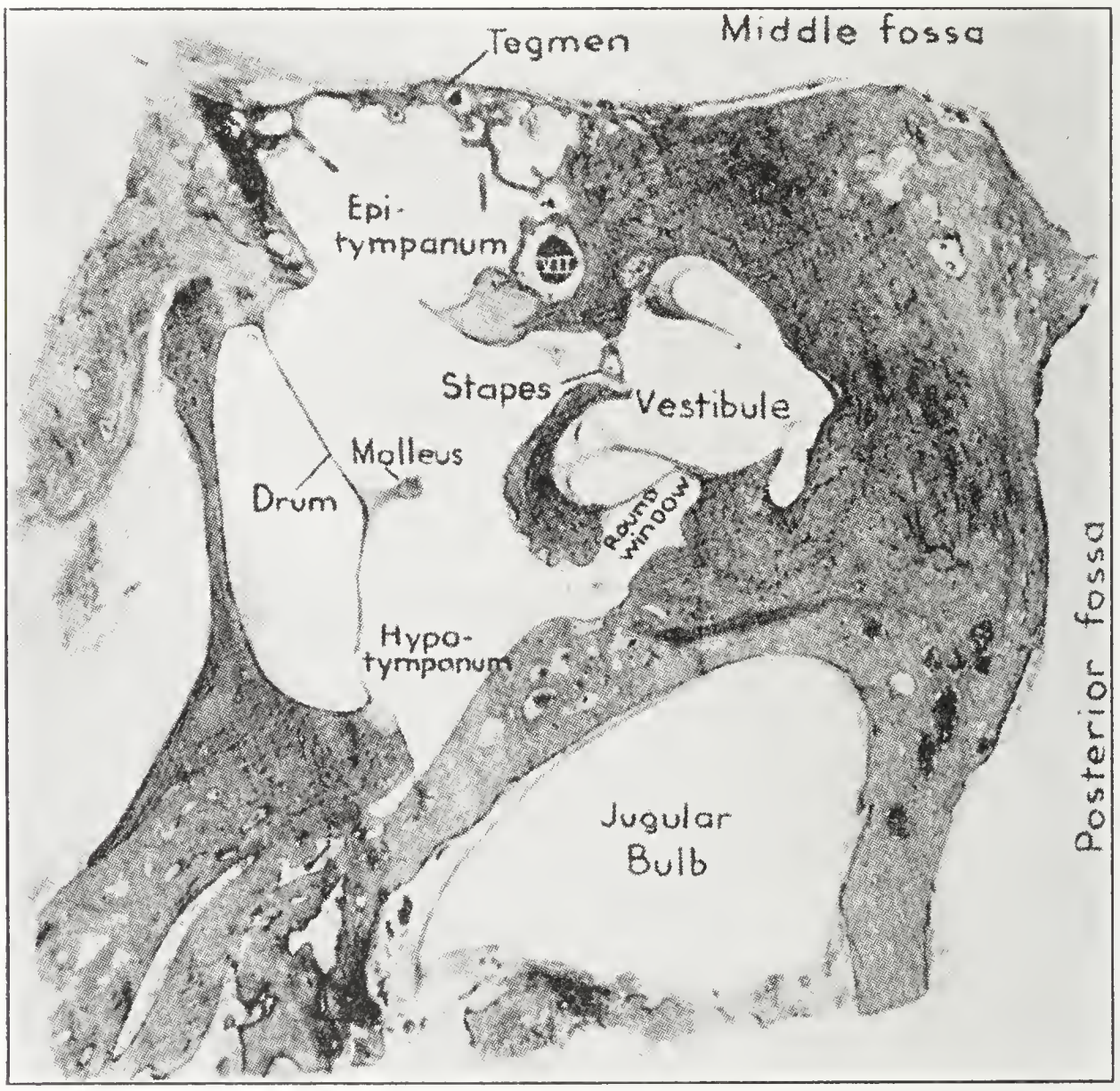

FIGURE 1.—Photomicrograph of Vertical Section through Middle Ear at Depth of Round and Oval Windows.

temporal bone is extensively pneumatized, there is more chance for infection to become shut off at a distant point where drainage through the middle ear is impossible. It is these cases which often come to mastoid surgery or to surgery deeper in the temporal bone.

Actually the middle ear is not a small cavity with specific dimensions as described in most text-books. It is impossible to measure the capacity of the middle without surgery. Whether it is the increase vascularization and local leucocytosis of inflammation which permits the body to take care of such poorly drained areas, or whether it is a stifling of the growth of the invading organism by the production of anaerobic conditions, has not been ascertained according to Fowler, but probably both these factors and more are responsible.

Mode of Infection

The complicated series of cavities in the temporal bone can become infected in a number of ways.

1. *In infants,* the most usual way is for the nasal secretions to run into the eustachian tubes as the child lies on his back during an upper respiratory infection. They cannot blow their noses or hawk mucus from the nasopharynx, and since their eustachian tubes are relatively short and straight, infection easily enters their middle ears.

2. *In older children and adults,* the commonest method of infecting the middle ear seems to be from blowing the nose too violently.

3. *Marked sudden changes in pressure,* such as those produced by deep diving may also suck infected material from the nasopharynx into the middle ear.

4. *A perforated or ruptured ear drum,* permits infection from the external canal to enter the middle ear, but here the usual cause of the infection is the nasopharynx. The perforation in the drum makes it very much easier to blow infected material into the ear because there is no longer a closed cavity.

Pathology of Otitis Media

This may be divided into four steps—

1. Edema of the mucoperiosteum and serous exudation.

2. Increased vascularization of the submucosa, infiltration with leucocytes, proliferation of fibrolasts and increased purulency in the exudation.

3. After two or three weeks, necrosis of the soft tissues as well as the bony walls of the cavity (shown by the X-ray) with this breaking down of the bone, the cortex of the temporal bone may erode at any point, producing an abscess behind the auricle, over the lateral sinus, in one of the cranial fossae, or even in the neck.

4. If the process is less fulminating, there may develop polypoid degeneration of the mucoperiosteum and a prolonged low grade osteitis and osteomyelitis.

Any one of these four processes may

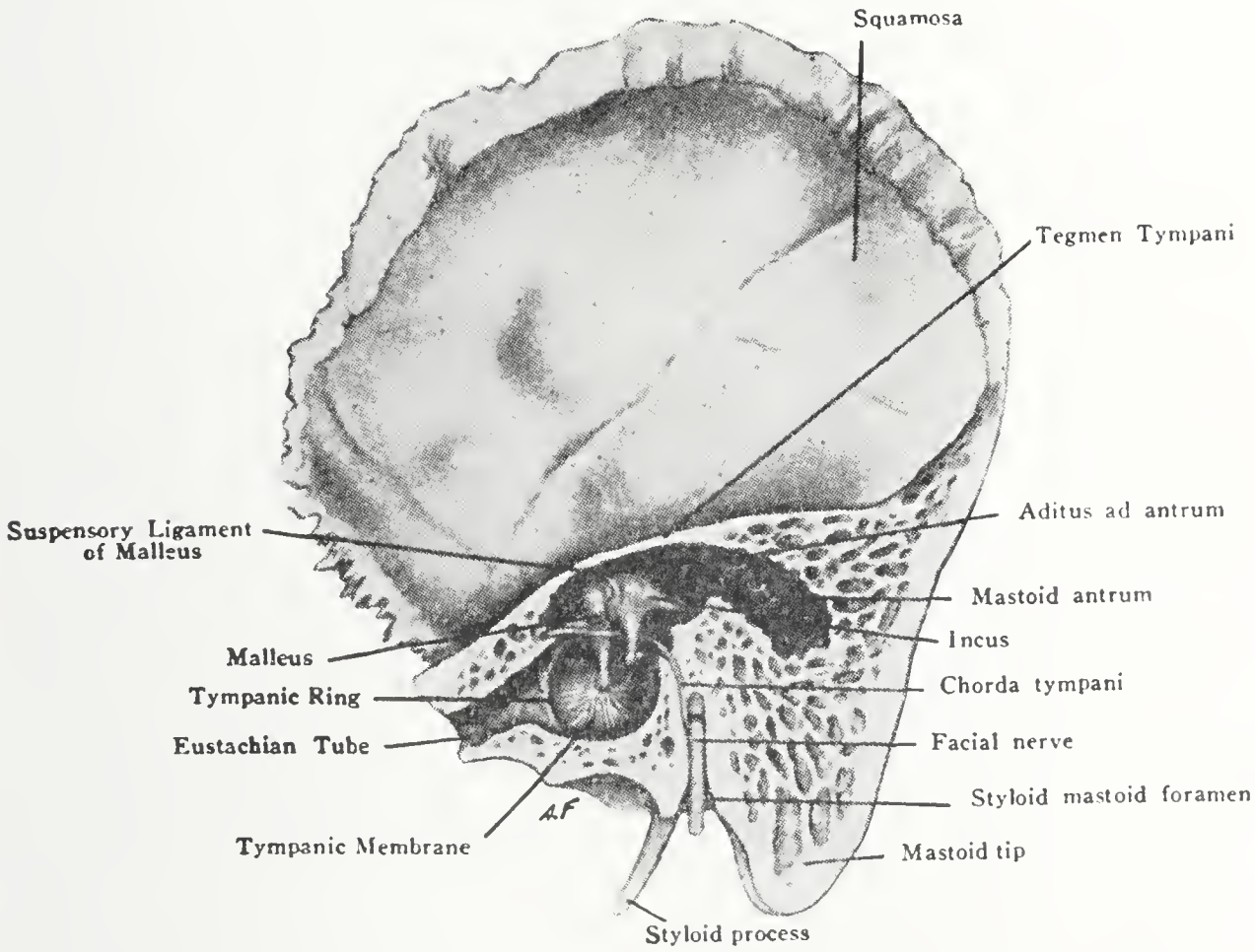

FIGURE 2.—Drawing from Wet Temporal Bone Specimen Showing Outer Wall of Middle Ear and Its Relation to the Tegmen Tympani and Mastoid Antrum.

stop, regress, and heal, leaving a more fibrotic mucous membrane and organized bands of scar tissue attached to the ossicles. Repeated infections produce similar processes in each instance but with the regrowth of bone in the previous infections the connection between the mastoid cells as well as the antrum is likely to be narrowed. This increases the possibility of infection becoming trapped deep within the bone, hence complications are more prone to occur in cases with a history of previous otitis media.

Clinical Picture

This disease begins as a non-suppurative otitis media so that at first the history and physical findings are similar in the two conditions, i.e., there is fullness and usually pain in the ear, with redness and then bulging of the drum with loss of landmarks and loss of air conduction hearing. There is often, in addition, a tenderness and sometimes edema of the mastoid region, and occasional edema of the external auditory canal. The drum becomes more and more inflamed as the disease progresses, and may rupture spontaneously, with alleviation of the pain, before a physician is consulted.

The discharge, either through these spontaneous perforations or from myringotomy is thicker and more apt to be hemorrhagic than in non-suppurative otitis media. If the disease has been present for some time, an idea as to the etiologic agent can sometimes be obtained by gross examination of the fluid. There are some observers who feel that they can diagnose the type of invading organism by the appearance of the drum and the discharge. This is extremely difficult and hazardous, but broadly speaking, a thin, profuse hemorrhagic discharge at myringotomy is likely to be infected with hemolytic streptococci; a yellow, purulent discharge is apt to be due to the staphylococcus aureus, and less profuse, thick, grayish discharge is apt to be due to the pneumococcus type III. The pain with hemolytic streptococcus is almost as severe as with staphylococcus, while in pneumococcus III (Streptococcus mucosus) the process is often so slow that, especially in adults, there may be no pain whatsoever. The lack of reaction in cases of inflammation of the middle ear and mastoid due to pneumococcus III—less bulging of the drum, less temperature, and elevation of the blood count—is the probable reason for the large number of complications which occur in cases infected with this organism. If no culture has been taken to give a note of warning, the patient and physician are lulled into complacency by the lack of symptoms until the insidious infection breaks through the cortex of the bone to invade the meninges, the lateral sinus, or the neck. With hemolytic streptococcus and staphylococcus, the temperature is a more reliable sign of the severity of the disease. The temperature in adults usually runs between 99 and 101 F., while in children it runs from 101 to 105 F. *High temperature indicates that drainage from the middle ear or surrounding spaces is inadequate.*

A leucocyte count is advisable as an index to the reaction of the patient. Leucocytosis runs high, especially in children, usually varying between 15,000 and 30,000 with 80 to 90 per cent. polymorphonuclear neutrophils, of which many are young forms. The erythrocyte sedimentation rate is also usually high, varying from 40 mm. per hour in mild cases to 160 mm. per hour in severe cases. Hearing tests are of little value in acute otitis media. In fulminating cases, it is sometimes necessary to test for bone conduction. A loss of bone conduction or a total loss of air conduction indicates that the middle ear infection has entered the labyrinth. In making tests of hearing, it is essential to provide adequate masking for the untested ear.

The only disease which is likely to be confused with suppurative otitis media is *acute external otitis*. In both there may be excruciating pain in the ear and deafness. In both there may be a purulent discharge. Temperature is not apt to be high in external otitis and tenderness on manipulation of the auricle is rarely severe with suppurative otitis media. There are more likely to be some inflamed lymph glands, either in the neck just below the auricle, or over the mastoid process simulating mastoid tenderness due

to mastoiditis, or over the parotid in external otitis. With acute external otitis there is often a history of chronic itching of the external canal or even "eczema" of the external canal.

The greatest difficulty in differential diagnosis of these two diseases comes when it is suspected that an acute otitis media or a chronic otitis media is present which has been the cause of the acute external otitis. Roentgenograms of the mastoid showing edema of the mastoid cells helps in the diagnosis of disease of the middle ear, but, as with non-suppurative otitis, clouding of the cells does not, as a rule, indicate an operative mastoiditis; in fact, all cases of suppurative otitis media show some clouding and this may last for many weeks after all symptoms have disappeared. This is especially so in petrositis. On the other hand, if *there is definite breaking down of cell walls* operation is, as a rule, imperative.

Types of Infection

Based on a series of cases at Columbia Medical Center in the years 1933 to 1939 it was revealed that specific chemotherapy is only applicable to less than half of the total number of cases of otitis media entering that hospital.

1. Over 35% of the cases were due to the staphylococcus.

2. More than 15% were due to organisms for which no specific chemotherapy has as yet been developed.

3. 30% were due to the hemolytic streptococcus for which we have sulfanilamide.

4. 12 to 16% were due to pneumococci for which we now have sulfapyridine.

From a study of these observations it was revealed that three-quarters of the pneumococcus and streptococcus cases get well within three weeks without chemotherapy, and that the *group that needs to be treated with chemotherapy is definitely a small one.*

Treatment

The treatment of otitis media for the last several decades has been directed towards the provision of adequate drainage for the middle ear and its surrounding air cells, somewhat as follows—

1. *If the infection was mild* (so-called acute catarrhal otitis media) drainage was usually obtained by reduction of infected material in the nose and by shrinkage of the eustachian tube.

2. *If the infection was more acute or more purulent,* incision of the drum was resorted to, after myringotomy or spontaneous puncture, the canal was simply kept clean to promote drainage with dry wipes or gentle irrigation.

3. *If these did not suffice* and the mastoid was seen to be breaking down in the x-ray, a mastoidectomy was performed, usually in the third or fourth week of the infection.

Since the advent of sulfanilamide a great many cases show subsidence of symptoms after chemotherapy so that many physicians have come to believe that it is the only treatment for otitis media, and now are using it in every case. The bad results from improperly conducted chemotherapy often reach the hands of the otologist. It can be said with certainty that the use of these drugs mask symptomatology, sometimes cause unpleasant reactions, and make the problems of management harder. We have, then, on one side the general doctor and the pediatrician who tend to treat all types of otitis media with chemotherapy, and on the other, the otologist who uses it only in desperate cases as meningitis. There must be some rule which has a scientific rationale. The rule can be built out of a study of the normal course of the disease, the pathology outlined and the results obtained in various types of cases by various methods of administration.

I previously stated that the group that needs to be treated with chemotherapy is a small one. How can we determine this small percentage? Lockwood *et al.* have definitely shown that sulfanilamide is less efficient in necrotic lesions, and that the more the necrosis, the less the likelihood that the drug will be sufficiently bacteriostatic to permit the body to annihilate it. I stated above, that very little necrosis (as a rule) occurs in otitis media and mastoiditis, until the second week of the

disease. It would seem, therefore, that in simple otitis media one can wait a week or ten days after the onset using the old, simple, mechanical methods of treatment. Then if there is no improvement and the case looks as if it might go on to surgical mastoiditis or a long siege of drainage, the administration of drugs may be begun with every expectation that they will stop the process if given in sufficient dosage. At the end of a week adequate bacteriological and blood studies should be available so that the correct drug can be selected. Analysis of cases treated with sulfanilamide shows that a large percentage of cases treated with adequate dosage, especially those in hospital, get well while those treated with small or short dosage in the clinic show no more tendency to recover than an untreated series. *The cases in which the drug has no effect are usually cases of inadequate dosage.* Often there is recurrence if adequate dosage is not prolonged well after the temperature is normal and drainage has stopped. If the drug is started late in the disease it may have no appreciable effect. Apparently the drug has less effect in cases where there has been a previous infection.

The physician must decide at the end of a week or ten days whether his case is going on to a surgical mastoiditis or is going to prolong itself enough so that the patient is in danger of acquiring a residual deafness. If he is fairly certain of one of these things chemotherapy should be instituted as a major procedure, comparable in every way to surgical interference. If possible, the patient should be hospitalized, and if not, careful blood and urine studies should be done every two days at home. The dosage should be fairly large (Long and Bliss suggest at least 2 mg. per kilo of body weight) and kept up for several days after the temperature has come to normal and the discharge behind the drum has ceased. If this is not done there will be almost certainly a recurrence of the disease, epsecially when the original organism is a virulent one.

Analysis of successful sulfanilamide-treated cases shows a dramatic subsidence of symptoms within three or four days. Most of them have no pain or fever after two days. This suggests that if there is no effect within three or four days the drug is not the answer, and that drainage will have to be instituted by one of the well-known surgical methods. In advanced or neglected cases surgery is usually essential, but of course may be supplemented by chemotherapy. After all, it is impossible to drain every nook and cranny in the temporal bone, and the chemotherapy will produce bacteriostasis in these unreachable regions. The combined use of surgery and chemotherapy is especially important in the complications of otitis media such as meningitis, brain abscess, and inflammation of the walls of the venous channels draining the temporal bone.

To recapitulate, given a case of otitis media, chemotherapy should be instigated a week or ten days after the onset of the disease, if the patient does not seem to be getting well without it and a specific drug is available to combat the bacteria of the infection. The chemotherapy must be given in adequate dosage for a considerable period after symptoms begin to disappear and under careful surveillance of reactions and with surgical adjuncts to the therapy in mind. In no case should it be given casually, without cultures and without a knowledge that most otitis media is self-limiting. It must be remembered that the use of bacteriostatic drugs makes the management of the cases harder because the symptomatology of the disease processes is masked.

ISLET CELL TUMOUR OF PANCREAS

By R. L. STIRRETT, M.D.

The case study to be reported is one of hyperinsulinism due to tumour of the pancreas. Harris postulated this syndrome in 1923. Such a condition was first diagnosed by Wilder and Mayo in 1927. Since this time a number of cases have been reported. The syndrome is now a definite entity and, quoting Whipple, a diagnosis of tumour of the pancreas can be made from the following triad of symptoms, namely:

1. Attacks of insulin shock coming on in a fasting state.
2. Hypoglycaemia below 60 mgm. % during an attack.
3. An attack relieved immediately by the administration of glucose.

The case report following is interesting in that it is not covered by the classical triad. It also presents several clinical features which merit emphasis.

The case was that of a married woman, 42 years old, who was perfectly well until about April of this year, when she felt headaches which were present on occasion during the following four months. Nothing further is known about these headaches. There were apparently no other symptoms. It seems unlikely that they had anything to do with the present state and we may date the onset of her present illness to August 3rd, 1940. On this day she had an atack in which she was mentally confused and wandered about the house aimlessly and ordered the weekend supply of bread twice—an unusual procedure for a thrifty housewife. She recovered from this attack, but the next morning, Sunday, she remained in bed in a semi-stuporous condition in spite of the fact that she was expecting guests. By 10 a.m. she was perfectly well, although she had had no food. On Monday she was given a thorough medical examination, but nothing was found. During this week she had two more attacks of mental confusion which cleared up spontaneously. No details of these are available. On August 9th she was found lying in bed in a stupor and she remained that way all day. The examination that evening revealed only right lower facial weakness, the reflexes were normal and there was no papilloedema. The patient had been incontinent by bowel and bladder several times during the day. During the examination the patient roused somewhat, but her articulation was poor and in naming test objects she called them all by the same name. Lumbar puncture was attempted unsuccessfully due to resistance on the part of the patient. Shortly after the struggle induced by the attempted puncture she suddenly regained all her faculties. At 3.00 in the afternoon of August 13th, one week after the beginning of her present illness she was admitted to the hospital, the story obtained being complete coma for the past day and a half.

Physical examination showed a large, obese woman, who was flushed, breathing stertorously with her eyes half opened. Further examination was negative, except that there was no response to stimuli and all her reflexes were absent. Examination of spinal fluid was negative. Blood N.P.N. was 58 mgm. %. As a pancreatic islet cell tumour was suspected, a blood sugar determination was done. The level at that time was 43 mgm.%. On receiving this report, she was given 100 c.c. of 50% glucose in addition to some intravenous 5% glucose which she had already been receiving. At 9 p.m. her condition was essentially the same clinically, though her blood sugar had by this time risen to 172 mgm.

The next morning, August 14th, her fever was less, but she was still comatose. Her blood sugar had fallen to 66 mgm. She again received 100 c.c. of 50% glucose intravenously, but remained unconscious, and her blood sugar that evening was only 42 mgm. On August 15th her blood sugar was 40 mgm. Her temperature was up to 104 degrees. She was still in coma. After several previous attempts had failed, duodenal tube was finally passed and the patient was fed. Blood

sugar that evening was 93 mgm., but there was no improvement in the woman's condition. Up to this time a diagnosis of islet cell tumour and hyperinsulinism had been considered likely, but in view of the failure of intravenous glucose to rouse her from coma, although the blood sugar was brought up to normal, the idea was abandoned. It was felt that the patient might be suffering from encephalitis, as during the last epidemic it was not uncommon to obtain a history of transitory bouts of confusion, later followed by coma with normal cerebrospinal fluids as found in this case, and intravenous sucrose was administered.

On August 17th the cerebrospinal fluid sugar was 73 mgm.—normal 45 to 80. The patient's condition remained unchanged until August 19th when her temperature quickly rose to 106 degrees, her pulse to 140, and she died 16 days after the onset of her present illness, five days after the onset of continuous coma.

Autopsy Report

At autopsy the only findings of particular interest were present in the pancreas and in the intracranial cavity. The pancreas was normal in configuration, except that in the tail 3 cms. from the end there was a small spherical nodule. Serial section of the pancreas showed no other abnormality. Assay of the insulin in the tumour found in the tail was found to be 30 units per gram, or approximately 15 times that of a normal pancreas. Microscopic examination showed typical adenoma of the islet tissue of the pancreas.

In the brain there was considerable congestion of the anterior cerebral veins. The cut surface showed some oedema with narrowing of the ventricles. There was, however, no visible cortical atrophy and no other abnormalities were seen. Microscopic studies of the brain revealed oedema and congestion of the medulla, midbrain and frontal lobe cortex. In the cortex of the post-central gyrus in the left frontal area, in the pons and in the hypothalamus there was subpial gliosis and there was also subependymal gliosis of the floor of the fourth ventricle.

Discussion

A typical case report, as taken from the literature, usually shows a history lasting from two to eight years. The history is of (1) *vasomotor symptoms*, (2) *psychic disturbances* and (3) *motor disturbances*. The *vasomotor symptoms* resemble those of sympathetic nervous system stimulation, namely: flushing, rapid heart, dry mouth. Concurrent with these symptoms there is an elevation of the blood sugar. It is thought that this blood sugar raising mechanism may be one of the body's methods of combating the hypoglycaemia. The release of adrenalin metabolizes the glycogen from the liver and so raises the blood sugar. It will be recalled that the patient under discussion was semi-comatose on attempting to do a lumbar puncture. After a short struggle she regained her faculties. It is thought that the struggle caused the release of adrenalin, which, in metabolizing liver glycogen, restored her blood sugar to normal limits.

Psychic disturbances commonly met with may consist of vulgarity, disorientation, loss of memory, aphasia, dysarthria, carelessness in dress and appearance.

The *motor disturbances* manifest themselves as pyramidal or extra-pyramidal lesions, temporary hemiplegia, marked Babinskis, incoördination, tremors and rigidity may be part of this bizarre picture. One Swedish writer states that these people present a picture very much like that seen in encephalitis.

The involvement of the nervous system is said to be caused by the inability of neurones to utilize the oxygen in the presence of large amounts of insulin and so a state of asphyxia exists. Apparently this reaction is reversible for a short length of time, so that relief of hyperinsulinism relieves asphyxia. In other cases, the reaction is not reversible, when death of the neurone occurs with consequent degeneration and gliolformation.

Observation on the effect of hyperinsulinism has further progressed since the introduction of shock treatment of Sachel for schizophrenia and other forms of insanity. It is usual to give some forty treatments to a patient. The patient is placed in coma for one and a half hours

by the administration of insulin. They are brought out of coma by the administration of glucose through a stomach tube. Workers in this field have classified coma as:

1. *Immediate*. Those relieved by dextrose given through a stomach tube.

2. *Delayed*. Those not relieved by dextrose given by stomach tube, but relieved by intravenous glucose.

3. *Prolonged*. Those receiving glucose by both the above routes, but which remain in coma minutes, hours to days.

One writer states that their records show one prolonged coma in every 1800 shocks. These cases of prolonged coma, depending upon the degree of hypoglycaemia or hyperinsulinism and the length of time for which the coma persists, usually terminate in death or leave the patient with residual nervous manifestations.

Hyperinsulinism in diabetes has been studied—one case is reported of a physician called to see a known diabetic in coma. He immediately administered 50 units of insulin. When the coma did not abate, he administered 100 units of insulin. As her coma became deeper and her breathing more stertorous, he felt that possibly a urinalysis was indicated. On finding no sugar he administered glucose. The patient lived and was later admitted to a Michigan State Mental Hospital as a mental defective. When later she died, autopsy showed cortical damage and gliosis.

One of the cases of pancreatic tumour reported by Doctors Graham, Robinson and Campbell several years ago, and who on admission had marked nervous system damage, has returned for observation and re-education. She is attempting to learn to walked again and shows marked dysarthria, aphasia and mental instability.

In conclusion, those using insulin for shock therapy and in diabetes have found that irreparable damage to the nervous system can result from hyperinsulinism; some cases of pancreatic tumour, even when removed successfully, still are left with residual effects. The patient under study with a short history of two weeks, lacking the long history of transient attacks of insulin shock relieved by food, underwent such severe damage from hyperinsulinism that intravenous glucose was not effective in controlling her coma, so that to the triad of symptoms characteristic of tumour of the pancreas formulated by Whipple, Harris and others, namely:

1. Attacks of insulin shock in a spastic state.
2. Blood sugars below 60 mgms. during attacks.
3. Attacks relieved by food

there might be added a fourth qualification stating that coma may persist even with a normal blood sugar in cases where hyperinsulinism has been severe.

Clinical Teaching in Roman Times

"I lay ill; but soon SyMMachus sought me
With a class of a hundred young men
Whose hundred cold paws have bought me
The fever I lacked till then."

"Martial and Medicine"
Dr. Raymond Crawfurd.

A PROBLEM IN DIAGNOSIS

By H. T. McCLINTOCK, M.D.
From the Medical Service, Toronto Western Hospital

Editor's Note: The Meds-at-Home is Jan. 31st. Let's have a lot of good answers to this problem so a ticket may be given away. All letters must be in to 107 Anatomy Bldg. by Jan. 25th, and addressed to the "Problem in Diagnosis Committee."

This history is that of a 56-year-old English female who was admitted to the Toronto Western Hospital on November 25, 1940, with following complaints:

1. Severe headache—3 days.

2. Flashes of light in visual field and decreasing vision—2 days.

3. Nausea and vomiting—1 day.

This patient was in good health until Dec. 1939 when she contracted pneumonia and following that developed an empyema and was ill with this until June 1940. During July she felt fairly well except that she belched gas in large amounts apparently not related to the type of food eaten and suffered from lower thoracic back pain. She was seen in Medical O.P.D. here on Oct. 21st, 1940. At that time examination revealed an apprehensive, emaciated female with a heart rate of 138 and a B.P. of 140/100 and no other positive findings. She was sent to a convalescent hospital following that and improved considerably in general health, gaining 6 pounds in the first 3 weeks. The belching of gas decreased considerably and she had somewhat less back pain, though it never disappeared. Three days before admission to this hospital developed severe generalized headache and increased pain between the shoulders, low down. Appetite became poor and she felt weak. Two days before admission she began to see flashes of light before her eyes and in the intervals saw black spots and suffered from dizziness. The same day she noted that her vision was becoming poor in both eyes and it became progressively worse rapidly. On the day before admission in addition to the above symptoms she became nauseated and vomited frequently. Examination at the convalescent home showed a B.P. 170/110 and urine contained albumin +++. with the result that she was transferred for investigation.

Functional Enquiry
Family History
Personal History
Past Illnesses
} Irrelevant.

Physical Examination

Temp. 99° (R). Pulse 98. Respn. 30/min.

A very thin nervous female with staring eyes. Answers questions in a disinterested manner and not always intelligently.

Head and Neck: Essentially negative except for the eyes. Vision is decreased bilaterally to light perception. Pupils round, regular, equal, 3 mm. in diameter and react to light. Ophthalmoscopic examination revealed evidence of some arterial sclerosis and some venous congestion. Occular movements can be carried out.

Chest: Essentially negative except for some scoliosis to left in the lower thoracic region with compensatory curves of spine.

Cardiovascular System: Heart rate 98—regular. B.P. 160/110. Apex beat—9 cms. to left of mid-line in 5th interspace. Mitral systolic and pulmonary systolic murmurs present. Sounds present in all areas.

Abdomen: Essentially negative.

Skin,—
Glandular System
Rectal Examination
} *No abnormalities.*

Back and Extremities: Scoliosis as described above. Some tenderness to percussion, slight pain over lower and dorsal spines on movement, but no notable limitation of movement, although patient com-

plains of poorly localized pain in lower thoracic region. Extremities not abnormal.

Nervous System: Within normal limits except for mental state as above and optic nerve changes.

Progress

Patient remained very restless and day after admission had back pain, and continued to vomit small amounts—no blood or food present. Three days after admission she became drowsy and confused, and towards evening was difficult to rouse. The following day at 1.00 p.m. she roused and asked for a drink. In a few minutes after this there was a sudden onset of stertorous respiration, cyanosis developed and 5 minutes later respiration became slow and shallow, ceasing after another 10 minutes in spite of coramine. With onset of dyspnoea complained of increased back pain and right shoulder tip pain.

Laboratory Examination

A. Blood.

Hb. 80%
R.B.C. 5.5
W.B.C. 7.2

Film essentially normal but a tendency toward hypochromasia.

B. Urine.

Neutral in reaction. Specific Gravity 1022. Albumin ++. Occasional hyaline cast and 3-4 pus cells per high power field.

C. Blood chemistry.

N.P.N. Nov. 26—49.
Nov. 28—67.

Blood Wasserman—negative.

D. Lumbar Puncture.

Pressure 160—Slight Xanthochromia and 5.6 red cells.

E. X-ray of spine.

Scoliosis as clinically evident. In addition in the lateral view there is slightly increased concavity of the anterior surfaces of 9th to 11th dorsal vertebrae.

The Natural History of Disease

"A half-century ago, the profession had acquired but imperfect knowledge of the natural history of diseases—that is, the history of diseases pursuing their course unaffected by active measures of treatment. The importance of this knowledge was not fully appreciated. Diseases which we now know to be self-limited in duration, and to tend intrinsically to recovery, were treated as if their continuance were indefinite and their course interminable, irrespective of active therapeutic measures. The influence on therapeutics of the knowledge of the natural history of diseases, acquired within the last fifty years, has been great. It is evident that this knowledge is the true point of departure for estimating the effect of treatment, and that without this knowledge the conclusions derived from clinical experience must often be erroneous. There is still room for the increase of this knowledge, and in this direction we are to look for further influence on therapeutics. With the now prevailing views, it seems a surprising statement that phthisis pulmonalis may be self-limited and end in recovery purely from an intrinsic tendency; yet, I venture the assertion that the correctness of the statement has been substantiated, and that before many years elapse it will be generally acknowledged to be a clinical fact. How important is this fact in judging of the agency of medicinal, climatic and hygienic measures in the treatment of this disease!"

Medicine of the Future
An address prepared for the Annual
Meeting of the British Medical Association.
By Austin Flint (Sr.), M.D., LL.D.
D. Appleton & Co., Bond St., 1886.

ACUTE PERIPHERAL ARTERY OCCLUSION BY EMBOLUS

By MARGARET ALEXANDER, B.A., '41

This article will include a discussion of the pathology, diagnosis and rationale of various treatments of embolic occlusion of the peripheral arteries. Special reference will be made to the brilliant experimental and clinical work done by Dr. G. D. W. Murray and Dr. Best. These contributions have thrown a new light on the treatment and a new hope in the prognosis of this condition. Even though successful embolectomies can be performed, and Heparin widens the field of usefulness and success of such operation, a diagnosis must first be made accurately and quickly. The onus of the diagnosis depends on the general practitioner. In any form of treatment the time element is all important. The subsequent fate of the patient's limb may depend on the doctor's ability to recognize the condition.

Pathology

An embolus, responsible for acute occlusion of a peripheral artery, most commonly originates as a mural thrombus in the left auricle or left ventricle. In the auricle, it is usually the result of rheumatic heart disease with mitral stenosis or degenerative heart disease with fibrillation. Not infrequently an embolus has occurred after normal rhythm was re-established, suggesting that a clot formed in the left auricle was expelled by the more vigorous contraction of normal rhythm. If the embolus originates in the left ventricle, it usually results from thrombus formation on an infarct produced by coronary occlusion: Less commonly an embolus arises from thrombi developing in an aneurism or on the surface of a rough atheromatous aorta. Vegetations in acute or subacute bacterial endocarditis may prove to be the source of an embolus in a peripheral artery. A very interesting and equally rare source of an arterial embolus is the so-called Paradoxical embolus. It originates from a thrombus in the systemic veins which passes through a patent foramen ovale into the left heart and thence into the arterial system.

From any one of these points of origin the embolus is swept off by the blood stream and carried to a point in the arterial system. It tends to lodge in an artery at a point of rapid diminution in size. The block therefore tends to occur at one of the main divisions of the peripheral artery. This is an important point to remember when one attempts to localize the embolus. It occurs more commonly in the arteries of the leg than the arm.

From this point, the fate of the part supplied by the occluded vessel depends upon the collateral circulation which in turn depends upon the state of the vessels, the strength of the heart and efficiency of the general circulation, and the consistency of the clot.

If a collateral circulation is efficient, the anastomotic vessels undergo active dilatation and the ischaemic part is supplied with blood before much damage is produced. The walls of the dilated vessels become thickened to accommodate increased pressure. It is obvious that arteriosclerotic vessels and a failing circulation would be unable to meet the increased demands and maintain sufficient circulation to the peripheral parts. It is also obvious that a soft young embolus may break up when it reaches a bifurcation and form small emboli which descend along the terminal branches and that this type would be more likely to produce gangrene than a firmly organized old embolus that lodges firmly, remains intact and leaves terminal branches open.

When an embolus lodges in an artery, the arteries supplied by the occluded vessel are thrown into spasm. This spasm lasts from 24-48 hours and if extreme may render inefficient a collateral circulation that would otherwise be adequate. It is for this reason that antispasmodic drugs have proved to be of benefit in many cases.

When an embolus lodges in an artery, the intima adjacent to the embolus under-

goes a change in 6-10 hours. Therefore it was found before the use of Heparin that embolectomy was successful only if performed early, because even though the embolus was successfully removed, a thrombus would immediately follow at the site of the injured intima. Thrombus formation often occurred at the site of removal or the suture line in a patient weakened by chronic heart disease and accounted for failures in some of the earlier embolectomies.

Effect of Heparin on Thrombosis

Heparin was discovered by Howell and purified by Best, Charles and Scott. It is prepared in the Connaught labs from ox lung. It is available in 10 c.c. vials containing 1000 units per c.c. One vial costs $5.00.

Howell's original idea was that Heparin would prevent the clotting of blood. It intercepts the clotting process either as an antithrombosis or an antiprothrombia. There is considerable evidence in Dr. Best's work that it will also prevent thrombosis. He has prepared a moving picture of his experiment which demonstrates the effect of Heparin, in sufficient quantity to affect the clotting time, in preventing the accumulation of platelets on a scratch in a glass slide. He showed that when the Heparin was removed and the clotting time returned to normal the platelets again accumulated on the scratch. How Heparin prevents thrombosis is unknown, but it has been suggested from his experimental work that it may have a specific effect on the platelets themselves. Whether it affects the stickiness of the cell so that it doesn't adhere to the injured surface or affects the cell membrane so that it doesn't break up and liberate thromboplastic substances, hasn't been decided.

With experimental proof that Heparin would prevent thrombosis, Dr. Murray experimented to see if Heparin would keep the lumen of the vessel patent after embolectomy. His experiments with dogs produced very satisfactory evidence that Heparin would be of value in operations on peripheral blood vessels.

In 400 clinical patients, he found that the vein used for injection never became thrombosed. On several occasions the injection was continued 10-14 days and the needle was removed only because of skin reactions. This affords clinical evidence of the prevention of thrombosis.

Dr. Murray reported 12 successful arterial embolectomies in his Hunterian Lecture. He found that if the lumen were completely cleared of the plug and the circulation restored, all the cases were successful when treated with Heparin. He introduced the drug into the vessel at the time of operation and the patient was heparinized (clotting time kept above 20 min.) for 3-14 days. In all cases the vessel remained patent and circulation has been satisfactory.

He had the opportunity of examining at autopsy an artery upon which he had operated 25 hours after the onset of symptoms and he found the vessel patent. From this he concludes that many embolectomies which were formerly doomed to failure because of late operation, can be successful if treated with Heparin. He also concludes that in his experience with the use of Heparin in embolectomy, many more cases will be spared amputation.

Diagnosis

The pain and paralysis that an embolus occasions when it lodges in a peripheral artery alarms the patient and he is obliged to seek medical aid without delay. The signs and symptoms attending this condition are very clear cut.

Complete occlusion is characterized by the lightning onset of severe pain at the site of the occlusion. This pain, which is of maximum severity at the onset, is of short duration and is probably due to spasm at the site of the embolus. It is followed very shortly by numbness and later coldness. These last two symptoms begin peripherally and extend up to a point which is a little distal to the level of the block. After a while, the original pain subsides and is replaced by prolonged distress or actual pain in the area of numbness and coldness. This late pain is due to ischaemia.

Partial occlusion is responsible for a sensation of tingling or numbness and

tingling and is to be compared with the numbness of complete occlusion. When the sensation of numbness is followed by the sensation of tingling, it is a clinical manifestation of re-established blood flow to the ischaemic part.

If a sudden acute pain occurs in the distal part of a limb after the occlusion of a more proximal artery, it probably indicates that an embolus has been carried from the original point of occlusion and become impacted further down.

Physical examination:

1. Limb is waxy white or shows a blotchy cyanosis.
2. Decreased skin temperature.
3. Partial or complete anaesthesia of skin.
4. Absence of tendon reflexes.
5. Absence of pulsation.
6. Failure of voluntary movement.

Localization

In localizing the site of impaction, several points should be borne in mind. It must be remembered that the evidence of ischaemia will not extend as far proximally as the site of the embolus since the branches arising proximal to the occlusion will supply the superficial structures for a variable space. If one recalls that an embolus tends to lodge in one of the main divisions of the peripheral arteries one is guided by the site of the sudden acute pain and the position of the first bifurcation of the artery above the ischaemic level.

If the vessel is superficial, one can determine by palpation the exact point where pulsation ceases. One may feel at this point a slight enlargement of the vessel.

The oscillatory method proves to be a very adequate routine method of determining the location of an embolus. Localization in this manner is accomplished by applying an ordinary blood pressure cuff at various levels of the ischaemic limb. The indicator needle shows definite oscillation above and little or none below the point of obstruction.

The use of X-ray with thorotrast has been suggested and used. The accuracy of this procedure is not to be doubted, but the dangers involved in its use do not warrant its routine adoption.

Differential Diagnosis

It is a difficult task at times to distinguish between acute occlusion due to arterial embolism and that due to arterial thrombosis. If embolectomy is to be considered this distinction must be made. A differential diagnosis is made possible by a careful study of the sequence of development of signs and symptoms. History of initial pain at the level of the occlusion doesn't occur in spontaneous thrombosis. On the other hand, thrombosis is characterized by a gradual onset of numbness or numbness and tingling. In the lower extremities, one usually finds a history of intermittent claudication, numbness and tingling, and coldness, leading up to the symptoms of acute occlusion. Since thrombosis occurs in arteriosclerotic vessels, it is rare under 50 years of age, whereas embolus occurs at any age. In thrombosis, the pain occurring distal to the occlusion is like the late or ischaemic pain of embolism.

Treatment

The principle aim in treatment of this condition is to re-establish adequate circulation in the ischaemic part, although it is evident that the success of any form of local treatment will be determined ultimately by the severity of the primary disease.

Principles of treatment:

1. Early diagnosis before the results of deprivation of blood supply become too marked.
2. Render collateral circulation more efficient by abolishing the spasm of the arteries supplied by the occluded vessel and by the use of passive vascular exercise as provided by the Pavaex machine.
3. Protect the ischaemic part from immoderate heat or from further reduction in its blood supply by elevation.
4. Embolectomy.
5. Arteriectomy.

Surgical Treatment

I. Embolectomy.

Any type of operation that is performed mainly on the victims of severe heart disease is doomed to carry a high mortality. But it seems reasonable to assume that if the clot is completely removed, if the use of Heparin will keep the incised vessel patent post operatively, and if the operation is performed before ischaemia has done too much damage, the patient may be assured of immediate recovery of the local condition. The primary condition remains unchanged regardless of the type of local treatment. If we regard such a procedure in the light of the number of cases of gangrene, amputation and even death that it prevents, it would seem to be the ideal treatment of acute occlusion due to embolism in lower limbs within the first 10 hours. Embolectomy combined with the use of Heparin marks a great advance in the field of modern surgery. Heparin is introduced into the vessel at the time of operation and the patient is heparinized (clotting time kept above 20 minutes) for three to fourteen days following. Heparin is used in a 2% solution although if there is an indication to reduce fluids it may be given in a more concentrated solution. Continuous intravenous drip is used. It is introduced into the leg at first but the patient is encouraged to move about in bed. It is then introduced into the arm and the patient should be ambulatory a couple of days before the Heparin is discontinued.

II. Arteriectomy.

Leriche believes that this procedure decreases the increased vasoconstrictor tone of the smaller vessels of the collateral circulation. This operation is not practised here.

Conservative

In many cases embolectomy is impossible. At any rate conservative treatment should be adopted for the *immediate* treatment of all patients with acute arterial occlusion.

1. Rest in bed must be insisted upon. The patient should be kept warm by the use of a cradle or heat to the entire body. Heat should not be applied directly over the affected limb.

2. Papaverine Hydrochloride anti-spasmodic 1/3-1/2 gr. given intravenously every four hours. This drug is effective in one half an hour.

3. Some look hopefully on alternate suction and pressure of pavaex. This is ideal in the treatment of early cases. It is a very effective method of treatment. Hermann claims that the chief source of failure is inadequate treatment. He recommends 5 hours per day for 3 weeks.

As soon as diagnosis is made a patient should receive intravenous injection of ½ gr. of Papaverine Hydrochloride and should be transferred to a hospital where he will be treated by Papaverine every 4-6 hours, for three or four days. Pavaex and warmth. In the embolic occlusions of the lower limb embolectomy must be considered as ideal if seen early.

Because of the better collateral circulation of the upper limb, Papaverine and warmth are probably the only methods of treatment necessary.

In cases where embolism is seen late Pavaex heat and Papaverine are indicated.

"Medical Aspect of Bayonet Charges"—

"By the way, one hears in novels and sometimes in history of bayonet charges—of cold steel. I never saw a bayonet wound, and of 25,000 wounds in Grant's battles, including the hand-to-hand struggle of the 'Bloody Angle', there were in all fourteen bayonet wounds; there were probably as many men severely kicked by mules!"

—The Medical Department in the Civil War.
S. Weir Mitchell, M.D.

THE OPHTHALMOSCOPE AND THE FUNDUS OF THE EYE

By P. R. BLAHEY, B.A., '41

PART I

Helmholz on introducing the opthalmoscope in 1851 writes, "I believe that alterations of the vitreous body and of the retina which, until now, have been found in cadavers, will also permit a recognition in the living eye—a possibility which appears to promise the most remarkable advances for the hitherto undeveloped pathology of the structure." The clinical observations, however, outstripped the accumulated observations in pathological specimens. The proper assessment of the new observations was impossible, for the gap between clinical facts and theoretical interpretations grew immense. To a student of medicine it is a strange "uncharted sea" of confusing descriptive terminology and shattered theories.

The limited objective of this article is but to aid the student in the interpretation of the findings as he sees them through a small "peep hole" of the ophthalmoscope. To do this it is necessary to have a working knowledge of the ophthalmoscope and the uses it may be put to. Finally the common abnormalities which are so frequently seen, will be discussed and where possible their clinical significance will be given. The retinal pictures with which the student should make himself familiar are not many. The chief are neuritis, papilloedema, arteriosclerosis, haemorrhages, vascular spasm and occlusion, and the various forms of retinitis. Perhaps the greatest difficulty is in retinitis, in which the etiology is rather varied.

In most cases the appearance of the eye grounds will at least give a clue to the possible cause or causes and the case can then be investigated along other lines. An obvious advantage is that ophthalmoscopic examination will sometimes give early warning of the onset of serious systemic or local disease. The conditions of the fundus will often, also, give a line as to progress and treatment.

Preparation for Examination of Eye

An adopted routine which will save time and produce good results is suggested. Each patient should first be examined without a mydriatic, the tension of the globes tested and where necessary a second examination is made after the pupils are dilated. In conjunction with ophthalmoscopic examination of the eye one can not overstress the necessity of obtaining a history for the cases with reference to vision, to test the eye reflexes, visual acuity and to examine the extent of the visual field, with special reference to its periphery, the size of the physiological blind spot and the presence of scotomata.

Macular vision may best be tested by having the patient look directly at the examiner's eye and then placing the head of a small pin directly in line with the pupil of the patient. This vision is compared with that of the more peripheral region, where lower acuity is normal.

Procedure of Examination

A dark room is preferable but not essential, since much information may be gathered otherwise. Similarly a mydriatic is not essential where a sufficient part of the fundus may be seen to rule out any possibility of abnormalities. An approach which will reduce the possibility of error, is suggested. The beam of light from the ophthalmoscope is first directed across the cornea from the region of the external canthus, to see if there are any opacities in front of the iris, such as fine corneal scars or central nebulae, which cause marked visual loss, and blur the observer's view. Dots on the corneal endothelium (Keratitis precipitates) that have a triangular arrangement in the lower central part of the cornea, indicate inflammation of the iris, ciliary body, or uvea. They are seen as flecks, which are white when they reflect the light, or black against the red reflex of the fundus of the eye.

The patient is next instructed to look straight ahead, and fix his gaze on some distant object. The examiner then, holding the ophthalmoscope a number of centimetres from the patient's eye, and fixing his own gaze at infinity, looks through the pupil with the instrument in the zero position. The so-called "red reflex", unless obscured by corneal, lenticular or vitreous opacities, is seen. The instrument is then brought close to the subject's eye, whereupon the nerve head and vessels are clearly visible. If the structures examined are not in focus, the graduated minus or plus lenses are tried in turn until the one which provides the clearest view of the fundus is found. The strength of the lens affording the best view provides a rough index of the error of refraction: a plus lens indicates farsightedness, and a minus lens, near-sightedness. Different areas of fundus are examined routinely, paying special attention to the disc, the blood vessels and the macular region. Since the ophthalmoscope magnifies sixteen times, the normally transparent retina is observed at the range of a low-power microscope and enables the recognition of such small structures as dilated capillaries. A gentle side to side movement of the observer may serve as an aid in interpreting the presence of swellings, tumours, or vitreous opacities, by the movement noticed of the superimposed objects.

Opacities of the lens are routinely examined with a plus 6.00 or plus 8.00 sphere. Opacities of the lens are always present in the fifth decade, and when central, cause distortion of the fundus picture which may erroneously be interpreted as a blurred disc margin, irregular vessels, or as a refractive error.

Normal Ophthalmoscopic Findings

(I) THE FUNDUS

The composite colour of the normal fundus is determined chiefly by the pigment layers of the retina and choroid, and to a less extent by the vascular layers of the choroid and retina. The range of this colouration varies from a chocolate of a negro to a yellowish red of the albino. Some races (Indian and Oriental) present a stippled fundus due to a less even deposit of pigment in the retinal pigment layer.

(II) DISK

The reddish white colour of the disc is produced by the non-medullated nerve fibres and their capillaries on a reflecting background, the lamina cribrosa. Normally the nasal side of the disc is redder, more prominent and has a more indistinct margin. The physiological cup is formed by the separation of the non-medullated nerve fibres in their distribution. It is whiter than the rest of the disc and at the bottom there may be seen a small area with greyish spots which are due to meshes of the lamina cribrosa. A white scleral or a dark choroidal crescent or ring may be noted along the margin of the disc in cases where the respective layers of the eyeball are not covered by the retina.

(III) THE BLOOD VESSELS

The retinal artery, which is a terminal artery, emerges from the disc at its centre or slightly to its nasal side with the vein on its temporal side. Near the disc the nerve fibre layer may present an appearance of fine radiating lines. The artery usually divides into upper and lower branches, each of which later subdivides to supply the respective quadrant from which the names of the vessels are derived. The arteries as seen with the ophthalmoscope are straighter, narrower, and a brighter red, than the veins. Tortuosity of the vessels varies greatly in healthy eyes. On a histological and physiological basis the primary branches of the central retinal artery are true arteries and the smaller branches are true arterioles.

The macular region is supplied by many small vessels given off the secondary branches of that side and a few small vessels which run straight out from the temporal side of the disc. The inner portion of the retina, as far as the outer border of the inner nuclear layer, receives its nutrition from the retinal arterial tree. The remaining layers are dependent on the capillary network of the choroid.

(IV) THE MACULA

The position of the macula, though well known, is rarely recognized ophthalmoscopically. It should be remembered that, due to its sensitivity to light, it is best to examine this region as a last step in the routine.

(V) CONGENITAL ABNORMALITIES OF THE FUNDUS

1. *Coloboma* — Various degrees of hereditary faulty closure of the foetal ocular cleft are occasionally seen. These may be graded for purposes of description as:

1. A crescent of the optic disc formed by the altered distribution of nerves and/or a thinning of the retina at the line of the cleft, i.e. the lower part of the fundus where streaks of pigment become visible.

2. A colobomatous disc with a gleaming white dome shaped colomba fringed with a pigment along the margin of the cleft.

3. A complete coloboma involving the disc, retina, ciliary body and iris.

4. And finally a cystic protrusion from the lower part of the eye ball.

(2) *Medullated Nerve Fibres*: The nerve fibres usually lose their medullary sheafs just back of the lamina cribrosa. Abnormally they may retain their sheets and appear as a white striated tuft which has a feathery edge and most commonly is continuous with the disc, but may appear as an isolated patch. Retinal vessels if present in this white area are hidden by it.

(3) *Pigment Anomalies*: Varied and irregular deposition of pigment has been observed, and its essential feature is that it does not undergo any changes. The commonest abnormality is an aggregation of flat polygonal plaques of pigment, several of which may lie together and tend to resemble a pattern not unlike dirty paw marks of an animal.

(VI) TRAUMA

Violence to the eye most commonly causes haemorrhages or detachment of the retina. The haemorrhages produced may be of any form but are usually superficial. They clear up quickly and unless a deep central haemorrhage takes place there is no permanent loss of visual ocuity.

Photoretinitis: This is a form of trauma to the central area of the retina caused by exposure to bright light (probably infra-red burn). The central vision may be temporarily affected but in more serious cases there may develop a mottled pigmented area in the macular region with varying degrees of permanent decrease of vision.

(VII) HAEMORRHAGES

1. *Pre Retinal*: They are large bright red and due to gravity, have a convex lower border and a flat upper border. They may follow trauma or be spontaneous. In the young, idiopathic recurrent bilateral haemorrhages from the retinal arteries into the vitreous form a clinical entity known as Eal's disease. These haemorrhages are absorbed slowly and there is no residual damage.

2. *Superficial*: A superficial haemorrhage which takes place into the nerve fibre layer is typically flame shaped. They accompany chiefly toxic and obstructive states. They are absorbed slowly and do not affect vision.

3. *Deep Retinal*: These haemorrhages are typically circular, punctuate or rosette shaped and lie deeper in the retina. They are typical of low grade toxic states. They remain unchanged for a long time and may leave a small permanent opacity.

4. *Subretinal*: Haemorrhage into the subretinal area is usually a large circular, brownish or cloud like areas which occur mostly in the central region. It is particularly characteristic of degenerative states. They invariably organize to form a plaque which permanently affects central vision.

(VIII) THE BLOOD VESSELS

1. *The Light Streak*: Normally light is reflected from the convex cylindrical surface of the blood column and the surrounding structural layers of the vessel coats. The light streak on the arteries is wider and brighter than that of the vein of a similar calibre. It is considered,

therefore, that the outer surface of the muscle coat contributes the important factor to the light streak. A thickening of the muscle coat and an increase in the index of refraction of the medial tissue will cause an increase in the width and brightness of the arterial streak. This change is the most important sign of arterial sclerosis. The artery assumes a "Coppery" appearance. In extreme cases the vessel is of a "silver" whiteness which obscures the blood column.

2. *Visibility of Vessel Wall*: Under normal conditions the walls of the blood vessels, except for the major branches in the optic disc, are not visible. An opacity of the vessel wall either by fibrosis or lipoid infiltration will be seen as a white streak on either side of the blood column. This is seen in both atheroma and arteriolar sclerosis and is the earliest evidence of a diseased vessel wall. The thickened medial coat is seen due to its increased thickness and optical density without a loss of transparency. Atheromatous plaques present irregular calibre of the blood column, and an opaque whitish region adjacent to or overlying the blood column.

3. *Arterio Venous Crossing*: At the junction of the arterio venous crossing the vessels fuse so closely that the endothelium of the vein lies directly on the outer surface of the artery and a common adventitia surrounds both vessels. Any change of tortuosity, a thickening or displacement of the retinal artery will alter the angle at which the two cross and will affect the size of the lumen in the vein at the crossing. Displacement of the arterio-venous segment is seen in hypertensive cardio-vascular diseases, and in advanced stages of atheromatous disease of the aged. An arteriolosclerotic thickening reduces the space available for the vein. When the vein passes in front of a sclerosed artery, it arches forwards as a result of an increased separation between the blood channels of the two vessels. If the vein passes beneath an artery the same separation may be recognized but a more prominent feature is a partial venous stenosis with engorgement produced peripherally. In advanced atheroma the disease of the artery may spread to the adjacent vein with a corresponding narrowing of the venous lumen for some distance next to the artery. Rarely a complete venous occlusion is observed and the development of collateral channels is first noted as a localized capillary hyperaemia in the retina near the obstruction and later as cluster of dilated tortuous capillaries which bridge the obstruction.

4. *Spasm*: Spasm of the central retinal artery or its branches is transitory and often recurrent. The patient will complain of momentary "blacking out" of vision.

5. *Retinal Artery Pulsation*: The retinal artery is seen to pulsate when the diastolic pressure within the vessel is at or near the pressure of the surrounding tissue. The diastolic pressure of the retinal artery (40 mm. Hg.) is generally half of the systemic diastolic blood pressure. The normal ocular tension (18-20 mm. Hg.) is one-half of that of the retinal artery.

The retinal vessels may thus be observed to pulsate in a case where the intraocular pressure is increased to the level of the diastolic pressure of the artery (glaucoma) or where the diastolic pressure of the general circulation falls to the level where the pressure of the retinal artery is near that of the intraocular tension (aortic regurgitation). Thus if in glaucoma the intro-ocular tension reaches to or near 40 mm. Hg. or in aortic regurgitation the diastolic pressure of the systemic circulation is below 40 mm. Hg. pulsation of the retinal vessels may be expected.

6. *Tortuosity of Vessels*: The diagnosis of abnormal tortuosity of the arteries is based on the finding of vessels which weave in a plane vertical to the retina. This is noted when it is found necessary to change the lens in the peephole in order to keep the borders of the vessel in clear view. This inconsistant level of the vessels along its course indicated the presence of retinal oedema.

In arteriolar sclerosis the arteries also manifest a pathalogical tortuosity in a plane parallel to the surface of the retina. Instead of the normal serpentine tortu-

osity, the vessels commonly turn sharply at right angle bends. This accompanies structural changes in the wall of the vessel and when evident is considered pathognomonic of arteriolar sclerosis.

7. Obstruction of Central Retinal Artery: A visible and permanent damage to the retina is produced in a few hours by a lodged embolus, arterial thrombus or a long lasting spasm of the artery. The entire fundus becomes a whitish pale or cloudy with a characteristic cherry red spot at the macula where the choroidal circulation shows through the thin retina. The white clouded retina undergoes a coagulative neurosis. Within the blood vessels, which are collapsed, small red beads of blood may be seen to pass slowly in either direction. When a branch of the vessel is involved, similar signs are noted in the respective quadrant with a corresponding loss of the visual field.

In the majority of cases there is a residual loss of vision which accompanies complete atrophy of the retina. When the symptoms and signs are due to spasm of the central retinal artery, there is a marked and usually complete recovery of vision in several days after the passing of the spasm.

8. *Venous Thrombosis*: Thrombosis of the central vein usually takes place at the lamina cribrosa, while a branch thrombosis often commences at the site of pressure of a thickened overlying artery. The latter characteristically occurs in people past middle age who are subjects of arterial disease, usually sclerosed retinal vessels.

The thrombosed vein is engorged, black, turgid and tortuous. Retinal haemorrhages and exudates which take place chiefly in the nerve fibre layer are extensive and indicate serious retinal damage. In central vein thrombosis the disc is blurred by haemorrhages and exudates and may resemble papilloedema.

The visual field is not materially affected in branch thrombosis but if the temporal branch is involved, the macular vision is usually lost by the outpouring of blood. Clinically there is a gradual clearance of signs over a period of months with which there may be some improvement of central vision, if it is involved.

(IX) DISEASES OF THE BLOOD FORMING ORGANS

The so-called characteristic picture of the fundus in cases with blood dyscrasias are seen only in advanced forms of the disease:

1. *Anaemia*: Where the haemoglobin is at a low level the disc and fundus are characteristically pale. The vessels lose their identity and tend to resemble each other. Accompanying this small haemorrhages, cloudy areas of exudate may be seen.

2. *Leukemia*: A blurred disc with an anaemic fundus and engorged pale vessels paint the picture. When retinal haemorrhages are present they are usually superficial with a white centre of aggregated white blood cells. Often the veins show a broad white line of perivascular infiltration.

3. *Erythema*: Engorged retinal veins with small superficial haemorrhages at or near the disc are characteristic in advanced cases. This may closely simulate central vein thrombosis.

4. *Purpura*: Like any other part of the body, the retina may show haemorrhages which are usually of a superficial character as an indication of purpuric states or conditions producing diffuse petechial haemorrhages.

Several conditions of the retina are described and termed as a retinitis. In a strict sense, they are not inflammatory. In choroidal inflammation, or what is termed a choroido-retinitis, there is always an involvment of the retina in proportion to the choroidal damage. Vision is affected by the changes and effects produces by the disease on the retinal cells and fibres.

The lesions characteristic of retinitis are retinal haemorrhages, exudates and disturbances of the pigment.

1. *Haemorrhages*: These have already been discussed. They may take a superficial flame or diamond shaped form or a deep punctate more persistent character.

2. *Oedema*: Retinal oedema indicates the presence of an increased intracranial pressure, metabolic disease or retinal arteriolo-sclerosis. The retina characteristically is clouded and has a rippled light

reflex. The rippled appearance is due to an uneven surface as a result of the stretching of the transverse fibres of the retina. It is also noted that the oedema causes the vessels to become tortuous in a plane perpendicular to the surface of the retina.

3. *Exudates*: Exudates are characteristically ill-defined greyish or faded fluffly areas which, like haemorrhages, tend to hide the blood vessels or elevate them so that spheres of different strength are required in order to obtain a clear focus on the vessel wall at different sections of the field.

4. *Pigment Disturbances*: Disturbances of the pigment are characteristic of certain degenerative diseases of the retina. A general statement may be made that lesions of the retina or the choroid, whatever their nature may be, tend to cause pigmentary disturbances such as proliferation, aggregation, migration, or a complete disappearance.

(X) TUMORS

Tumors of the retina may be single or multiple and vary from what appears to be a well circumscribed light rounded area in the retina to any form of bizarre picture.

(XI) FOREIGN BODIES

With a history of trauma, the detection of a foreign body serves a useful purpose in prognosis and treatment.

(To follow in next issue)

ANSWER TO PROBLEM IN LAST ISSUE

Pathological Findings in a Case of Anuria

By F. H. Cote, B.A., M.D.

In the December issue a recent case of anuria was described and a few short remarks were made on anuria in general. The pathological findings in this case are now summarized briefly.

The case was that of a man of fifty-two who developed upper abdominal pain followed by diarrhoea and anuria five days before admission to hospital. Despite measures to promote a diuresis he passed fifty ounces of urine only in the ten days he was in hospital and he died clinically of anuria (NPN 275) and terminal bilateral bronchopneumonia.

At autopsy—performed ten hours after death—the only gross and microscopic changes demonstrable in the urinary tract were as follows. There was an acute nephrosis. Both kidneys were congested. There was a small cyst of the right kidney. There was cloudy swelling of both kidneys with haemorrhage into, and slight fatty degeneration of the renal tubules. The ureter, kidney pelves, bladder and prostate were congested and inflamed. There was a small abscess on the bladder wall, and a slight degree of hydroureter involving the lower portion of both ureters.

The other internal organs showed some congestion and oedema. There was oedema of the subarachnoid space and congestion of the brain. There was an acute bronchitis and bilateral bronchopneumonia involving the lower lobes. Both lower lobes were oedematous. There were numerous pleural adhesions, and a small amount of fluid in both pleural cavities. The heart was hypertrophied weighing 395 grams. The coronary arteries were normal. The stomach was congested. The spleen was certainly enlarged weighing 2370 grams. The liver was fatty with slight ascites approximately 100 c.c.'s. These were the only noteworthy pathological findings. The congestion of the brain, liver, slight hydrothorax, ascites, etc., is readily understood on the basis of the large fluid intake and minimal output of the patient while in hospital. It will be recalled clinically that there was no surface pitting oedema. The small abscess in the wall of the bladder may have been due to trauma from the catheter. There was no obstruction in the urinary tract. No definite cause for the nephrosis was demonstrated. There were no crystals in the tubules, and there was no history of the patient having taken such drugs as Dagenan or Sulfathiazol. One must conclude that something either in the history, or clinical finds was overlooked which may have shed some light on the cause of the anuria in this case.

Dr. F. H. Côté

STERILITY IN WOMEN DUE TO GONOCOCCAL INFECTION

By R. H. WESLEY, '42

Sterility, by definition, means barrenness or the inability to produce young. In a narrower sense, it is usually interpreted as meaning the inability to conceive young, and this latter definition will be followed here.

If conception does not take place after normal sexual intercourse the fault may be in one of three sources or in a combination of these. The man, or woman, may have defective germ plasm, or some factor may be impeding the passage of normal spermatozoa to a normal mature ovum. Gonorrhoea may be a cause of any or all of these problems, but it is in the reproductive organs of the female that it takes its greatest toll.

Statistics regarding the causes of sterility are voluminous and variable, but all agree in one respect, namely that gonorrhoea is the greatest single cause. The figures of Beclere and Francois of Paris are among the most recent but they are considerably lower than the normal estimations. Even their report showed 50% of cases were due to gonococcal infection.

Bacteriology

The gonococcus is a small Gram-negative diplococcus, having its two adjacent surfaces flattened or slightly concave, with a small oval interval between them. When seen in exudates and in the tissues it is largely intracellular in distribution, especially in the later stages of the disease. The gonococcus is a very strict parasite, being dependent for spread on direct transference from host to host, and man is the only animal which it naturally infects. The organism is very delicate and is difficult to culture. The most satisfactory medium for growth is agar containing unheated blood, serum or ascitic fluid. It requires plenty of oxygen in a moist atmosphere and grows best at a temperature of 36°C. The organism can be found in abundance during an acute infection but is often difficult to isolate when the active stage has passed.

Modes of Infection

Sexual contact is by far the most common and most important mode of infection. Contamination of the conjunctival sac of the newborn infant is another common method of transmission, and a severe non-venereal vulvovaginitis occasionally occurs in young girls in institutions due to the common use of utensils or clothes.

The gonococcus attacks chiefly the urethra, both in the male and female, producing an acute inflammation. The cocci quickly penetrate the surface, passing between the epithelial cells which are loosened and desquamated, and invade the tissues as far as the superficial layers of the submucous connective tissue. There is a profuse emigration of polymorphonuclear leucocytes and the discharge soon progresses from mucoid to purulent.

Almost simultaneously with the urethral infection the organism starts its invasion of the female genital tract. A surface inflammation is the first sign, but by direct continuity it gradually involves the deeper-lying tissues. Remissions and exacerbations are common, for under proper stimulation an infection, apparently quiescent, suddenly springs to life. This chronicity is the most outstanding characteristic of the disease and it is from this standpoint that it becomes of such great importance in sterility. Infection of the vulva, urethra and vagina yield readily to treatment, and here the disease leaves behind it no barrier to subsequent conception, but with invasion of the cervix, body of the uterus or oviducts, the eventual course is quite different, and sterility is a common complication when these structures have passed through a marked attack of gonorrhoea. Infection of Bartholin's and Skene's glands may be likewise a factor in sterility by causing dyspareunia.

Bartholin's (*Vulvovaginal*) glands and Skene's ducts (*lying just within the urethral meatus*) are almost always infected when a woman contracts gonorrhoea. Palpable enlargement of Bartho-

lin's glands is due to gonococcus in most cases and may be evidence of a cyst or, less commonly, an abscess. An abscess of the gland causes acute swelling and severe pain and frequently produces temporary sterility by prohibiting coitus as being too painful. These abscesses may rupture spontaneously and pour forth a purulent discharge; but recurrence is frequent and the condition rarely disappears until the affected gland is excised during a quiescent period. Infection of Skene's ducts also causes sterility from the resultant dyspareunia, but prompt treatment readily relieves the condition. Some authorities go so far as to say that thickening of Skene's ducts and a discharge of pus from them is pathognomonic of a gonococcal infection.

The vagina is not often attacked and due to the secretion of lactic acid, the absence of glands and the thick resistant stratified squamous epithelium, it frequently acts as an efficient barrier to the progress of the disease above this level.

Infection of the Cervix

Gonorrhoea of the cervix is an extremely frequent condition and unless promptly treated may lead to sterility. Stricture of the cervix is a fairly rare sequel to gonococcal infection, and this, combined with a purulent mucous plug, is the chief hindrance to the passage of the spermatozoa, although the lumen itself may still be quite large enough to admit them. It has been suggested that there is a specific enzyme in human spermatozoa which will dissolve this plug of mucous by lysis, but its activity is diminished or stopped by the presence of pus in the mucus.

Infection of the cervix, usually specific, is one of the causes of acquired stenosis. When this is found in young women there is usually a history of genital infection, as a result of which a marked inflammatory reaction of the cervical tissues is produced with oedema and hyperplasia. There is always a certain amount of permanent thickening of the cervix in cases of chronic inflammation, and this is frequently increased by too active local treatment and manipulation. As a result the opposed cervical mucous folds are often injured and subsequently fusion may take place, or, due to a gradual increase in connective tissue and scarring, the canal becomes severely constricted. A badly diseased cervix of this type often produces sterility especially if a purulent leukorrhoea is present as is commonly the case.

A cervical mucous plug and purulent cervical secretions are commonly removed by topical applications such as douches, tampons, cauterization and dilatation. If the cervix is severely diseased, and the genitalia are otherwise healthy, surgical removal of the affected cervical tissues usually removes the sterility. Specific treatment, directed against the gonococcus itself and applied before the disease becomes too far advanced, is most efficacious. This consists of therapy by selected members of the sulphonamide group.

Disease of the Uterus

When extension of the disease from the cervix to the endometrium or body of the uterus takes place, it is usually at a menstrual period or shortly postpartum when the secretions are alkaline and hence not inhibitory to the gonococcus. Chronic endometritis, with a thickening of the lining membrane of the uterus, acts as a very rare cause of sterility by interfering with the proper imbedding and growth of the fertilized ovum. The disease, when present, spreads to the underlying metrium, the uterus is enlarged and soft, depending on the severity of the infection, and its walls are friable and easily torn. However, the condition is not often present without infection of the tubes and is overshadowed by this much more serious and common menace.

It must be remembered, also, that conditions in the uterus are constantly changing and the infection is usually thrown off, or progresses to the tubes, before it is detected clinically.

Disease of the Fallopian Tubes

It is in the Fallopian tubes that gonorrhoeal disease of the female produces its most serious effects. Infection of the other organs is temporarily a cause of

sterility, but when the tubes become infected the disease pursues a rapid and damaging course and all too frequently terminates in complete occlusion of the tubes with subsequent permanent sterility. Tubal disease does not usually occur until two or three weeks after the initial onset of the disease, and then, although nearly always bilateral, rarely attacks both tubes at the same time, usually infecting one several days or a week before the other. The exact method of infection of the tubes is not known, either direct continuity or retrograde menstruation may carry the infection upward. As already suggested the gonococcus ascends more rapidly during menstruation or postpartum.

Curtis estimates that 70 to 80% of cases of salpingitis are caused by gonorrhoea, but other writers give a higher estimation and only a few produce figures showing a lower incidence. All coats of the tubes are involved and the fimbriated ends become swollen, retracted and agglutinated. The ciliated epithelium lining the tubes becomes granular and desquamated in places; a purulent or serous exudate is present in the lumen and the mucous folds often become adherent to one another. The accumulation of an exudate is almost invariably in the outer, more dependent infundibular and ampullar portions, and as the uterine end frequently is also blocked, hydrosalpinx or pyosalpinx is produced. This occlusion of the fimbriated ends may be complicated by, or may be due to, adhesions between the fimbriae and the ovary or other adjacent surfaces. Peritubal adhesions cause the tube to be drawn down behind the broad ligament and to become functionless, and this is complicated by a localized pelvic peritonitis which occurs in many cases with adhesions to adjacent loops of bowel, omentum and peritoneum. Peritoneal adhesions are characteristic, but gonococcal adhesions of this type are always amenable to separation by blunt dissection; stubbornly adherent tissues are pathognomonic of additional complicating infection.

This attempt on the part of nature to prevent extension of the disease to the whole peritoneal cavity, thereby closing the tubes and forming dense adhesions, results only too often in sterility.

Gonococcus does not remain in the tubes longer than two weeks after the disappearance of fever and leucocytosis. Persistently active gonorrhoea of the tubes is therefore due to recurrence of infection rather than chronic infection. Reinfection may be from male carriers or from the lower genital tract due to traumatism, passage of douches or menstruation. This is of interest in the condition called "one-child sterility." It has been thought that latent disease may flare up at child-birth and, through tubal infection, prevent further pregnancies. The evidence, however, does not support this hypothesis, and one-child sterility occurs as a result of gonorrhoea only through reinfection from below following labour.

In some cases the diagnosis may be complicated by the absence of any history of illness or even infection and there may be no palpable masses or tenderness. The external genitala should be carefully examined for evidences of specific infection, and additional accuracy in diagnosis is obtained through tubal insufflation (*Rubin test*) or lipiodol injection with the addition of radiography.

The treatments of acute tubal infection should be essentially nonsurgical. Patency of the Fallopian tubes is tested, as mentioned above, with Rubin's transuterine insufflation of carbon dioxide, and this may become an active therapeutic measure by opening constricted tubes. The test should be done the week or ten days following menstruation, and if it shows that the tubes are occluded or seriously obstructed, injection of an opaque medium (*e.g. Lipiodol*) is warranted, followed by roentgenography to locate the site of the obstruction. Surgery is indicated in selected cases of sterility and for the drainage of an abscess; if repeated attacks of salpingitis are occurring immediate operation should be avoided.

Disease of the Ovaries

The ovaries, since they are encircled by the ampullar portion and the fimbriated extremity of the tubes, are especially prone to infection. This occurs, usually,

after the extrusion of the ovum when a freshly developing corpus luteum is open to invasion, and the infection then generally goes on to abscess formation. Coincident with this abscess formation there is a general inflammatory involvement of the entire structure of the ovary, leading in time to complete destruction of its function. Sometimes the ovaries are involved in a veil of adhesions which prevent the ovum after liberation from reaching the tubes, yet the inflammatory process which caused the adhesions may not have involved the tubes to the extent of completely closing their entrance. The tunica albuginea of the ovary may become so thickened from the inflammatory reaction that the maturing follicle is prevented from rupturing, with the resultant formation of a retention cyst. These cysts when they are numerous may entirely or in part destroy the organ. Such a diseased ovary is usually removed coincident to the removal of the tube.

Treatment

The treatment of the various conditions causing sterility has been briefly indicated when they were considered, but since much can be done to halt the progress of the disease before the involvement has threatened fertility, a short resume of the treatment of gonorrhoea will be included.

Before the advent of the sulphonamide preparations the treatment of gonococcal infections was more or less limited to rest, topical applications mainly consisting of silver salts, medicated douches and urinary antiseptics. Now, however, these methods are merely used when the newer drugs fail or as a supplement to them. Sulphanilamide has been used with great success in doses which are very high and are continued for short periods. The drug is administered every four hours and four doses are given daily. The following method has been used successfully during a single course of treatment. (*Cannell*)

80 grs. daily for 3 days.
60 grs. daily for 4 days.
40 grs. daily for 2 final days.

Each dose is accompanied by half the amount of sodium bicarbonate. The supplementary treatment varies but the following consists of fairly standard routine: potassium permanganate 1 : 4000 douches, topical applications of 40% silver nitrate to the cervix, and 50% silvol to the urethra. Rest in bed is still an important factor in the acute stage.

With the use of sulphanilamide the drug resistant cases were about 32% (*Batchelor*), but when dagenan (*Sulphapyridine*) was investigated only 6% failed to respond to treatment. The principle of dosage is similar to that used with the earlier drug but the results are much more encouraging and the drug is more universally tolerated. Administration in the following way is suggested.

60 grs. daily for 5 days.
40 grs. daily for 5 additional days.

If signs are persistent treatment can be continued for 4 more days. Relapse should be avoided by giving high initial doses, because ensuing courses of treatment rarely produce as good results.

Summary

1. Gonorrhoea is by far the most important single cause of sterility.

2. Occlusion of the tubes is the most important effect of gonorrhoea in the investigation of sterility.

3. Treatment should be aimed at preventing the spread of the disease to the Fallopian tubes. The best drug for treatment is sulphapyridine.

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WOUND SHOCK

By G. G. SMITH, B.A., V B. & M.
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Latta in 1795 is said to have first applied the term "shock" to shock-like phenomena where no vital structure has been injured. There are valid objections to the term shock. Cannon suggested the term, exemia. However, authors continue to use and deplore the term, shock.

Early hypotheses abound in hypothetical concepts as—draining of vital fluid, loss of animal and organic powers, destruction of the great nervous power, and the like.

There has been no agreement on a comprehensive definition. Moon defines wound shock as a circulatory deficiency, neither cardiac nor vasomotor in origin, characterized by decreased blood volume, decreased cardiac output and by increased concentration of blood. As we shall see this definition has certain disadvantages.

Theories of exhaustion, acapnia, fat embolism, and decreased alkali reserve have all been weighed in the balance and found wanting.

Observations on Shock

Shock following battle wounds, without injury to vital structures or serious loss of blood, was a major problem in the field hospitals and casualty clearing stations of the allied forces during the first World War. The British Medical Research Committee organized an investigation of the nature, mechanism and control of shock. The Committee consisted of men of recognized ability in their respective fields. No one attempted the solution of the problem as a whole. Cannon's monograph on traumatic shock, summarized and correlated the material.

Shock developed most frequently after small multiple wounds, compound or comminuted fractures associated with extensive laceration. The impact of projectiles travelling at high velocity was transmitted laterally producing wide areas of destruction with slight hemorrhage. Shock developed four to twenty-four hours after the receipt of injury. Hunger, fatigue, exposure, rough journeys and faulty immobilization of injured parts greatly enhanced the danger of death by circulatory failure. Surgical treatment and the administration of ether frequently precipitated shock.

The appearance and clinical symptoms of a patient in shock although of importance will not be mentioned here.

Traumatic Toxemia

As experiences in the treatment of wounded soldiers accumulated the conviction grew that absorption of products from injured tissues was a major factor in the production of circulatory deficiency.

Wallace reports the immediate improvement in the condition of wounded soldiers following amputation of mangled limbs and the excision of all injured tissue was so remarkable as to appear a direct sequel to the removal of damaged tissue.

Bayliss and Cannon produced shock in cats by mechanical trauma to the thighs. The previous severance of the lumbar cord did not prevent shock. Shock did not occur if vessels to the leg were ligated, but occurred after the release of these.

An injection of histamine produced shock. Plasma was lost, partly by leakage through capillary walls which had been rendered abnormally permeable by histamine, and partly by stasis of blood in diluted capillaries. Lewis believes that in extensive tissue damage, H-substance is released sufficiently to cause dilatation and permeability of capillaries in systemic areas as it does locally when released in small amounts. He believes this to be the method of wound shock production. It has been shown that injections of substances derived from animal tissues disturb the physiology of the capillaries. Dale and Krogh have shown that cells respond to injury by liberating substances whose effect on the minute vessels are like the effects of histamine.

Evidence against Traumatic Toxemia

The postmortem appearances of the tissues after histamine shock are different from those after trauma. Dragstedt and Mead could not demonstrate a histamine-like substance in the blood in shock from trauma. They were able to detect such a substance in anaphylactic shock. They showed that if histamine is injected in amounts which reproduce the effects following trauma, its presence can be demonstrated in the blood by their methods.

Local Fluid Loss

In 1927 Blalock did some experiments on dogs and stated it was impractical to distinguish hemorrhage from shock. His dogs were anaesthetized and one hind limb traumatized. After death the posterior parts were bisected with care and the weight of the traumatized and untraumatized halves compared. The difference in weight consisted of extravasated blood and serous fluids in and about the traumatized areas, and averaged 35% of the total blood volume.

The authors found:

1. No evidence of shock after release of obstructed circulation from traumatized areas.
2. Fluid from the normal and from traumatized areas caused a decrease in blood pressure.

They concluded shock was due to the local loss of fluid. They believed burn shock was caused by fluid lost in and about the burned area.

Evidence against Local Fluid Loss

Blalock admits that a vasodilator substance is liberated locally by cells as a response to injury. He maintains there is no convincing evidence that this hypothetical product causes a general capillary reaction. Moon (1940) injected trypan blue, an indicator of capillary permeability, and found the staining of the tissues not limited to the injured areas but that it involved visceral regions remote from the injury—indicating disordered capillary function in other areas.

In many instances the fluid loss at the site of injury is not sufficient in itself to account for the death of the experimental animal.

We must conclude that local fluid loss does not give us the whole picture.

Nervous Factor in Shock

There have been many who have claimed that a nervous factor was important in shock. However, the literature is contradictory. The role of the nervous mechanism, if one is present, cannot as yet be clearly stated.

Conclusion

In determining the mechanism of wound shock a consideration of cellular permeability is fundamental. It is inseparable from most cellular activities of life—from an invasion of the ovum by the sperm to the exit of the spirit, called death.

Moon strongly supports the toxemic theory. He believes that metabolic substances, liberated either by functional activity of cells or by injury to cells, cause dilatation and increased permeability of capillary endothelium. This leads to a vicious circle whose central feature is capillary atony. There are many things which tend to maintain the circle once initiated. He believes local fluid loss is one of the many contributing factors. He makes a distinction between shock and hemorrhage. However, it must be remembered that posthemorrhagic shock shows the typical picture of wound shock.

Blalock states that the mechanism of production of all types of shock is not the same. Shock resulting from hemorrhage, trauma, burns, and other causes leading to a rapid and severe dehydration, he includes in the hematogenic type. The central feature is a decreased blood volume and blood flow to tissues. Shock produced by agencies acting directly on the vessels, e.g. histamine, he includes in the vasogenic type. He maintains that local fluid loss is all important in the production of wound shock.

There are many factors that accentuate and maintain the vicious circle of wound shock once it has been initiated. Of all the factors that bring on this vicious

circle by far the most important is the local loss of fluid in and about the damaged area. However, in many cases there appears to be some other mechanism which comes into play. The toxemic and nervous factors may act as the hair necessary to break the camel's back.

Therapy of Wound Shock

All workers in this field agree that it is easier to prevent the onset of shock than to treat the fully developed condition. Delay in treatment is more conducive to shock than any other single factor. Treatment to be effective must be carried out at once. Consequently it is of the utmost importance to recognize impending shock.

The central theme of treatment is an early and adequate restoration of an effective blood volume. The restitution and maintenance of this volume in a patient whose capillaries have allowed and are continuing to allow a loss of all the plasma elements including protein, is very difficult.

Whole blood would at first appear to be the ideal answer to this problem. However, there are many disadvantages to its use. The correct donor must be obtained and at best the serological tests and preparations for transfusion are time consuming. Then again in those cases where there has been a greater loss of the fluid element, whole blood adds to the already concentrated blood producing increased viscosity. Despite great care numerous reactions are still being reported.

During the Spanish Civil War, blood banks were developed to a high degree of efficiency. Stored blood has a limited period of usefulness (5-10 days). The Peyton-Rous mixture prevents hemolysis, but there has been no preservative found that will prevent the escape of potassium from the cells into the plasma. The intravenous minimum lethal dose of potassium for man is not known. However, in shock where large amounts of fluid are administered rapidly it is wise to use comparatively fresh blood. Stored blood shows a decrease in platelets and a drop of 50% in the leucocytes during the first twenty-four hours.

These various drawbacks led to the search for suitable blood substitutes.

The use of saline and glucose infusions for the treatment of shock produce only temporary benefit. It appears that crystalloid solutions further dilute the plasma colloids to a point where it is impossible to hold the administered fluid in the blood stream.

During the Great War, gum acacia solutions were employed as a means of introducing colloid. Acacia is not a protein but it has the property of attracting and holding fluid in the blood stream. Studdiford (1937) reported severe and fatal reactions from its use.

Now in hemorrhage there is a reduction in blood volume by the actual loss of whole blood. In some types of shock there is the same diminished blood volume but a greater loss of plasma than the cellular elements. This condition demands the addition of protein to increase the colloidal osmotic pressure. It has been accomplished with fair success by the use of blood serum or plasma, or human ascitic fluid.

In shock produced by peritoneal cooling in dogs, Mahoney found the protein loss was almost entirely albumin. The intravenous injection of albumin alone is at present impossible. However, the use of plasma or serum has proved very satisfactory in the treatment of this condition. They supply the necessary proteins without increasing the viscosity of the blood. Mahoney, using plasma, observed that the ultimate results were the same as whole blood—but the elevation of the blood pressure was more prompt and the improvement in the general condition more striking. Shock from burns, operations in the peritoneal cavity, and gunshot wounds of the abdomen have been favourably treated with plasma.

On the other hand Best, Solandt and Clegg and Dible and numerous others have employed serum with equally gratifying results. It was Levinson who first emphasized the very important fact that serum produced beneficial results even in cases involving severe hemorrhage.

Plasma

Plasma can be preserved with merthiolate at room temperature for many days. If the temperature is reduced to 4°C. it can be kept for months. In the frozen state it can be kept longer. Finally reduced to a dry powder by desiccation, it can be preserved indefinitely and readily prepared for use by regeneration with distilled water.

The use of dried plasma simplifies storage and transportation and reduces bacterial growth. Group AB is the ideal plasma for transfusion. Since this is the rarest blood, a successful plasma has been prepared by mixing group A with B. This can be administered without cross matching. Large doses produce no reactions.

Serum

In May (1940) Best and Solandt used the Thalhimer technique to concentrate serum to 1/3 of its normal volume. They felt that any procedure useful in severe shock should be initiated by the administration of a vasoconstrictor, pituitrin, temporarily to correct vascular atony so that solutions introduced into the blood stream are not immediately lost into the tissue spaces. It was hoped this procedure would break the vicious circle responsible for vascular atony, and that adequate vascular tone thus initiated would be maintained after the effects of the vasonconstrictor had worn off. The concentrated serum was administered when the pressure was rising under the influence of the vasonconstrictor Shock produced by histamine, trauma with and without hemorrhage, was successfully treated by this method.

Magladery, Solandt and Best believe that in post hemorrhage shock if the blood pressure could be raised to 100 mm. Hg. without the use of a vasonconstrictor, a good end result was assured. They have concluded that it is questionable if permanent recovery can be expected in an animal in which the blood pressure has sunk so low as to require the use of a vasonconstrictor.

Serum can be stored in large amounts for a long time without harm. Type O serum can be given to any recipient. A mixed serum in which the various types are represented in the same proportions as they occur in the population is now being tested. Favourable results will greatly simplify serum administration.

Serum vs. Plasma

Several workers have reported troublesome reactions with serum and prefer plasma as a therapeutic agent. Ravdin and his associates (1939) cautioned against the use of lyophiled serum. Strumia, Wagner and Monaghan (1940) claim that serious reactions are not due to the lyophile process but to the use of serum. What gives rise to the difference between serum and plasma is not known. Best and Solandt have used serum and plasma interchangeably with identical results. The amount of fibrinogen in plasma has an insignificant effect on the osmotic pressure. Plasma and serum, free from reaction-producing substances, are physiologically and therapeutically identical.

Human Ascitic Fluid

Human ascitic fluid obtained from the peritoneal and pleural transudates has been used by Davis and White and found efficient in raising the blood pressure in secondary shock. It costs nothing, is sterile, contains proteins, and has an osmotic pressure comparable to that of blood. Choisser and Ramsey have been able to remove the agglutinins in the ascitic fluid, thus making cross-matching unnecessary. However, the problem of the low protein content of ascitic fluid has still to be solved.

Role of the Adrenals

Many observers have reported hyperactivity of the adrenals in shock. Adrenalectomized animals show extreme susceptibility to a wide range of relatively minor insults. In the urine of all persons who were in a state of potential shock, Weil found a cortin-like substance which would protect adrenalectomized rats from insults fatal to them but not to normal rats.

Perla and his associates have produced good results in patients undergoing severe surgical operations by the administration of desoxycorticosterone acetate

before and after the procedures. Selye and Dosne on the other hand believe pretreatment is of little value and maintain that corticosterone is effective in combating shock in rats, but desoxycorticosterone is ineffective.

If pretreatment did prove to be important it would be of little value in coping with shock on the battle field.

Summary

The general principles and types of therapy in wound shock have been considered. The employment of the inhalation of oxygen in the prevention and treatment of shock is certainly a logical step, if, as we proposed, the cause of death in shock is anoxia. We can only await a more extensive trial of this method before we can assess its value. Serum and plasma, as substitutes for whole blood, appear to be definitely beneficial. There is also an important place for the use of concentrated serum to attract fluid back into the blood stream. An evaluation of the role of the adrenal cortical hormone in shock is at present difficult. Finally, enthusiasm for the trial of new methods should not lead to the neglect of those which have been established by previous generations—the relief of pain, cold, fatigue, and the combating of bacterial infection.

The Value of Reading Pages from the Past

"This is one benefit which the Medical Student may derive from making himself acquainted with the various systems which have prevailed in different ages and countries, although now they bear no authority. To study the revolutions of medicine and how long each opinion bore sway, by what means it was deposed, and what succeeded and reigned in its stead, is on various accounts, expedient. However false ancient theories may be deemed, many of them highly ingenious; by endeavouring to comprehend them, the mind is habituated to investigation, and strengthened by exercise; we discover what led men of great sagacity into error; such discoveries serve as beacons to prevent us from steering the same course.

Amidst the froth of false theory, many useful hints and observations may be found; for it often happened that the practise was good, even when the reasoning was founded on false principles."

Medical Sketches:
in two parts
By John Moore, M.D.
Printed at Providence by Carter & Wilkinson
opposite the Market 1794.

Book Review

Well, the attempt to find a specifically medical book to review hasn't been very successful. Of those I've read lately the best is "Who Walks Alone" by Perry Burgess. It's a novel about a leper, and although it doesn't tell you much about leprosy, it's an interesting psychological study. In naive terms the question it presents is: if you suddenly found you had leprosy, what would you do?

Hugh Young's "A Surgeon's Autobiography" is somewhat dull reading, but interesting in so far as it is an autobiography written for the medical public rather than for the lay public. It has a lot to say about venereal disease during the Great War and a lot to say about g.-u. surgery as a specialty, and it has a chapter on Woodrow Wilson's last illness in which the author tells the story of how he treated acute urinary retention successfully by "masterly inactivity".

Hertzler's new book, "The Doctor and His Patients", is a lop-sided and (in spite of its candidness) basically puritanical tract on sex problems: a very disappointing sequel to his autobiography.

"Moses and Monotheism", Freud's posthumous work, I haven't read, but according to a friend who did read it and who, incidentally, is susceptible to Freudian theories, it is a study which interprets the crucifixion in particular, and martyrdom in general as sublimations of adolescent sexual frustration. It's provocative, apparently, but not illuminating, and like everything else that Freud has written it opens up new fields but doesn't conquer them.

The German "Medical Guide for Air Personnel", translated by Henderson and sold by the University Press at the exorbitant price of $1.00, is mildly interesting, but if you're going to read it, borrow it from the library for it isn't worth buying.

The one book that seemed worthy of having an evening spent on it, even though I found parts of it distinctly unintelligible, was "Today and Destiny", a sort of Reader's Digest synopsis of Spengler's "Decline of the West". This is a book that matters.

Spengler's basic theory is that human history is a repetitive cycle of Cultures. Each Culture develops out of the unceasing ebb and flow of history; it becomes a Civilization: it passes through the phases of feudalism and bourgeois capitalism to democracy: it passes from democracy to Caesarism: it proceeds from Caesarism to disintegration, and so returns to the unceasing ebb and flow of the historyless.

"Growing from primitive barter-centre to Culture-city and at last to world-city, it sacrifices first the blood and soul of its creators to the needs of its majestic evolution, and then the last flower of that growth to the spirit of Civilization. So, doomed, it moves on to final self-sestruction."

This is heady stuff, but it bears pondering.

Strangely, Spengler is not the pessimist that this quotation suggests. As a matter of fact he is not in the least interested in pessimism or optimism. He is merly out to paint a new picture of history, to take the place of the old picture (ours) which bases itself on the sociological interpretation of Darwinian evolution. *We* look upon history as a linear progression from cave-man to Utopia, a straight-line development, a steady ascent. Spengler sees it as a sort of wave motion. Out of the formlessness a Culture rises like a wave; it climbs and breaks; another wave follows. But between the waves—nothing.

The basis for his theory is formed by an analysis of other Cultures which preceded ours—the Chinese, the Egyptian, the Graeco-Roman, eight of them in all. Time and again he draws parallels, often convincing ones. He shows how capitalistic economics and industrialism eventually work themselves out, and having done so, collapse. He shows how music and art, and even science, progress to a point where they can progress no farther. He shows how at a certain stage in the existence of a Civilization true creation dies. We may not agree with all this, but we've

got to admit he has a case. After Wagner, no new music. After the Impressionists, no new painting. Perhaps it's true.

The interest of what he has to say lies of course in the light it throws on the future of *our* Culture. And in considering this we must remember that Spengler published "The Decline of the West" in 1917. At that time he prophesied the coming of Caesarism in Germany, after a period of unsuccessful democracy. He prophesied, too, the world-wide growth of a new spirit of nationalism. He prophesied the age of war with*out* war—what we have termed the "war of nerves". He prophesied a world war after that of 1914. He prophesied the failure of capitalism in all countries, including America. And all of these prophecies are prophecies no longer, but facts.

If the rest of what he foresaw is to come true, then for *our* Civilization, and for ourselves as idealists, the future is pretty dark and gloomy. Democracy will vanish. The world will pass through a cycle of wars. Quiet will follow, but it will be the quietness of non-existence. The world will return to the uncultured, uncivilized, and uncreative chaos of the Middle Ages. And eventually a new Culture will rise, to repeat the history of ours. It sounds despondent and deadly. But then so is the world we live in, at the moment. And the truth is seldom pleasant when it's seen for the first time.

Whether or not this *is* the truth is a matter for the individual to decide. Spengler presents only one point of view: a terrifyingly tenable one, but not necessarily a true one. But true or false it is a point of view we must consider when we try, as at some time or another all of us do try, to watch history and to decide what part in history we play.

There's much more in this book—much more still, I suppose, in the original. And I feel I'm not succeeding in the least in interpreting even this small amount. When you really grasp what he's driving at you get a strange sensation of the motion of human existence. As you stand and watch people, masses of them, you can feel the whole thing moving forward like a huge dynamic organism—and you a part of it. It's disgustingly mystic, I know: but not everything that's mystical is worthless.

The real fun comes when you find some of Spengler's concepts creeping into your personal philosophy: crashing into it, rather, for it's more like a bull in a china shop than anything insidious.

Perhaps you saw Sherwood's play "There Shall Be No Night". Some people liked it. It was really little more than a sentimental splurge for the uplift of democracy, freedom and "our side". If the choice lies between Sherwood sentimentality and Spenglerian mysticism, mysticism seems infinitely preferable.

Essentially, no such book as this could ever change the way we live. All it can do, at best, is make us see life differently. Sherwood looks at present times with a tear-stained, chin-trembling attitude. Spengler looks on them as parts of a greater whole, vital yet on the grand scale unfeeling, emotional only in detail. We too can see life this way, if we try; but we *feel* it our own way.

For my part the beautiful cosmic thoughts of the declining west disappear in the shadow of present tragedy. All I can hear is the wailing banshee howl of the air-raid sirens in the ten minute movie called "London Can Take It". God, what a sound. Followed by the heavy drone of aeroplanes. And then bombs falling.

Unbelievable as it seems, Spengler is solace in the face of this. He'd turn in his grave if he knew.

The book, if you still want to know after all this confused reviewing, is "Today and Destiny", edited by E. F. Dakin, and published by Alfred Knopf.

C. G. S.

Abstracts

R. L. MacMILLAN, Editor

Contributors D. J. Breithaupt
D. P. Bryce

The Clinical Picture of Tuberculosis in Children

EDITH M. LINCOLN, M.D.

American Journal of Diseases of Children, 60: *371, August 1940*

This short well illustrated article presents something of the author's experience in the diagnosis and course of tuberculosis in children. The similarity between tuberculosis and other infectious diseases is stressed, the author considering it similar to typhoid fever or meningococcal meningitis in having a known portal of entry and symptoms of invasion, as well as a period in which the causative organism circulates in the blood stream, possibly reaching all parts of the body. Complications may then develop by local spread or by metastasis. However, in tuberculosis only, the original lesions may remain active for years, thus greatly prolonging the possibility of complications.

The Primary Complex is defined as consisting of the primary focus in the lung,—usually just under the pleura in the best ventilated part, the regional lymph node lesion,—usually the hilar nodes, and the interfocal zone connecting the previous two. Following this, allergy is established, the tuberculin reaction becoming positive three to five weeks following exposure. The establishment of allergy may result in an increased perifocal infiltration around the primary focus.

The ultimate course depends on the number of tubercle bacilli and the amount of tuberculo-toxins present, and also partly on the individual reaction. The primary focus may heal by fibrosis and calcification, or may caseate and progress by contiguity or bronchogenic spread. The regional lymph nodes may be reduced to a mass of caseous tuberculosis, and give rise to symptoms of irritation or obstruction in the lung. There may also be a blood spread within one to six weeks.

The absence of physical signs or symptoms in many cases, and our lack of knowledge in the treatment of tuberculosis in childhood is emphasized.

D. J. B.

Expulsion of Haemolytic Streptococci by Nasopharyngeal Carriers

RONALD HARE, M.D.

Canadian Public Health Journal, 31: *539, November 1940*

This is the account of work done to see how many haemolytic Streptocicci are projected into the air by carriers on talking, coughing, etc. Blood agar plates were arranged in the saggital plane on the arc of a circle 12 inches from the mouth. Tonsilitis, Scarlet Fever, and "Common Cold" subjects were used in the convalescent stage.

The experiments were well controlled so that the number of colonies of haemolytic streptococci found probably closely represent the number of haemolytic streptococci that reached the surface of the agar plates.

Although many colonies of common mouth organisms were found, very few haemolytic streptococci were cultured. This had been suspected because of the results obtained by earlier workers. Those plates which did contain haemolytic streptococci colonies were almost entirely those below this horizontal level. This was so even with forceful coughing, and the plates were only 12 inches away.

Dr. Hare concludes that haemolytic streptococci are not carried from bed to bed by droplet infection, as the droplets containing these organisms in the cases

studied were only in the heavier droplets, which would be rapidly carried to the ground. It is suggested that these evaporate on the floor, and the infection is spread by their subsequent disturbances so that they float, dry, in the air. Thus in cases of haemolytic streptococcal infection, unnecessary agitation of the bedclothes should be forbidden, and wet dusters and oily methods of sweeping the floor should be used. In the prevention of wound and puerperal infections, the wearing of masks is most advisable, as one is often directly over the wound, and 7% of normal people are carriers. However, it is suggested that equally good results may be obtained by refraining from speaking or coughing in the neighbourhood of the patient, or in the operating theatre at any time.

D. J. B.

Infectious Mononucleosis

ALAN BERNSTEIN, M.D.

Medicine 19: *85, February 1940*

This is a scholarly and comprehensive discussion of Infectious Mononucleosis, or Glandular Fever as it is sometimes known. The extremely varied clinical picture is stressed, and the only characteristic sign is the monocytosis, which is usually over 60% with a white cell count of 8 to 20 thousand at some time. The cells are described as being probably ofo lymphocytic origin, and are rather mature in appearance. The Paul-Bunnell sheep cell agglutination reaction (previously described by Hanganutziu and Deicher) is considered to be significant if positive in dilutions of 1 : 160 or more. Of 65 cases only 8 had a titre of less than 1 : 160, but if the clinical picture is suggestive, a negative Paul-Bunnell does not preclude the diagnosis of Infectious Mononucleosis. Horse serum injections, or parenteral liver injections may give a falsely positive reaction. A falsely positive Wasserman reaction may also be present. Fever, glandular enlargement, or even an abnormal white cell count are not considered to necessarily contraindicate rehabilitation of the patient. There is much interesting material in this article.

D. J. B.

Impulsions—A Specific Disorder of the Behaviour of Children

BENDER and SCHILDER

Arch. of Neur. and Psychiatry, November 1940

Everyone is aware or has had experience with the peculiar impulsive actions of children, some of which are so peculiar as to excite immediate interest as to their true nature. In this paper several extremely interesting examples of this phenomenon are reviewed and discussed.

Such impulsions may be defined as cases in which specific ideas, interests and actions dominate the clinical picture and make social adaptation impossible. They have been found mainly in boys between the ages of 4 and 12.

They take their origin from the preoccupations of infants, but are always related to the family situation and are not direct expressions of the sexual, motor or aggressive drives. However, due to the lack of the full development of self-criticism and condemnation, they fall short of the obsessional character trends seen on the adult.

Such impulsion may be often suspected from the early history of an adult obsession neurosis.

D. P. B.

Neurogenic Factors in the Production of Acute Gastric Ulcer

BOLES and RIGGS

J.A.M.A. 115: *21*

Such a controversial aspect of an obscure subject should catch the student's eye immediately. The etiology of gastric ulcer is still not established.

In this article the relationship between stimulation of the vegetative hypothalamic centres and peripheral vasomotor disturbances is thoroughly reviewed.

It is stated at the outset that the so-called neurogenic ulcer is a focal expres-

sion of a sub-clinical generalized circulatory insufficiency.

Such circulatory change may be the result of intracranial disease such as tumour or increased pressure, stimulating the hypothalamic centres. On the other hand such a condition may result in the absence of intracranial disease. In these cases it is stated that as a result of general constitutional disease reacting upon general circulation, the centres may be again stimulated as a result of relative anoxia.

Thus the production of the acute gastric ulcer, resulting from local vasoconstriction of the mucosa, is intimately connected with circulatory changes in the whole body and in particular with any stimulating conditions existing relative to the sympathetic centres and nervous system.

D. P. B.

Carcinoma of Ectopic Testis

J. B. GILBERT and J. B. HAMILTON

Surg. Gyn. & Obs., December 1940

This article definitely shows by statistical methods that an ectopic testis is a predisposing factor in the development of carcinoma of the testis. 850 cases of carcinoma of the testic were analysed and some interesting figures emerged. Carcinoma of the testis is 48 times as common in individuals with ectopic testis than in normal. 95% of those with an ectopic testis who developed carcinoma developed it in the ectopic testis.

R. L. M.

Response of Plasma Prothrombin to Vitamin K Substitute Therapy in Cases of Hepatic Disease

ALLE and JULIAN

Arch. of Surg., December 1940

The liver appears to be the site of prothrombin formation. There are many reports of prothrombin deficiency in cases with liver damage where no biliary obstruction exists. In this paper the failure of 12 such cases to respond to vitamin K therapy is discussed. While there is a low prothrombin level associated with biliary obstruction the administration of vitamin K brings the prothrombin to normal in 24-36 hours. The authors believe the administration of vitamin K and the determining of the prothrombin response may be used as a therapeutic test to distinguish intrahepatic disease from obstruction of the bile ducts.

R. L. M.

Disappearance of the Tubercle in Pulmonary Tuberculosis

J. M. MacMILLAN

Amer. Rev. of Tub., December 1940

The author calls attention to a group of cases who have had pulmonary tuberculosis and die of pulmonary disease but at autopsy no active tubercles can be found. Findings are emphysema, bronchiectasis, fibrosis and calcification. He shows, using three examples, that a person can overcome the tubercular infection itself but may die because of the pathological changes left in the lungs by the old infection. Tuberculosis may be the etiological agent of emphysema and bronchiectasis.

R. L. M.

Ammonium Bicarbonate Secreted by Surgical Maggots Stimulates Healing in Purulent Wounds

WILLIAM ROBINSON

Am. J. Surg., 47: 111-115, Jan. 1940

The healing effect of living blowfly maggots introduced directly into chronic non-healing wounds has been ascribed to allantoin and to urea, both products of maggot catabolism. But since these are produced in minute quantity only search has revealed an excretion of considerable amounts of ammonium carbonate which is unstable and is rapidly transformed to bicarbonate.

After experimental trials on dogs, sterile solutions of ammonium bicarbonate (0.25—4%) were then used on patients afflicted with purulent con-

ditions—chronic osteomyelitis ulcers, chronic ulcers such as diabetic, varicose, arterial, abscesses, etc. Twelve cases are reported in detail—in all healing was complete in from ten days to two months.

In the maggot treatment of wounds a relation of alkalinity to healing was observed and the alkalinity resulting from application of ammonium bicarbonate solution is of the same order as that produced by maggots.

A. M. J. C.

The Treatment of Acute or Recent Pulmonary Abscess

GEORGE J. HEUER, M.D., F.A.C.S.
Dept. of Surgery, New York Hospital and Cornell University Medical College

1. Conservative Treatment;

A series of 55 cases is presented, which were initially treated conservatively, that is, by general measures, postural drainage, and in a few cases by bronchoscopy. Where progress appeared satisfactory, no further interference was offered. However, in 29 of these cases, it was sooner or later felt necessary to resort to surgical interference. Of these surgical cases, 19 are now well, while 10 died. Of the remaining 26, 16 recovered and are now well, 7 are known to be dead, and 3 are believed to be dead, as a result of complications of their abscesses.

Thus, of the 55 cases treated conservatively, surgery was eventually found necessary in 29, or 53 per cent. Of the total series, 35, or 61.5 per cent. recovered, while 38.5 per cent. died.

2. Surgical Drainage:

Two series of cases are presented, one in which surgery was employed in the treatment primarily, and the other where surgery was instituted after initial conservative treatment. Again considering only uncomplicated cases of solitary abscess, of 24 patients treated primarily by surgery, recovery or improvement occurred in 95.8 per cent, while 4.2 per cent died. While of 27 patients treated initially by conservative methods, followed later by surgery, recovery occurred in 70.4 per cent, and death in 29.6 per cent. Thus this series agrees fairly well with that quoted under conservative treatment.

The author discusses the figures presented, and concludes that earlier surgical intervention is indicated in many cases, having in regard the fact that the percentage recovery in cases treated initially by surgery is higher than in those treated conservatively, and that even considering only the cases treated surgically, the percentage recovery is higher the earlier the treatment is instituted. He does not suggest however that postural drainage is of no use, but rather that it be used in conjunction with surgery. It is suggested that in no case should conservative treatment alone be continued for longer than one month in the absence of adequate response.

G. E. W.

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